BUREAU OF HYGIENE AND TROPICAL DISEASES.

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## PELLAGRA.

VISWALINGAM (A.). **Food and Disease in Malaya with Observations on Pellagra and Keratomalacia.**—*Malayan Med. Jl.* 1929. June. Vol. 4. No. 2. pp. 65–69. [12 refs.]

The extent to which ill health and disease in Malaya is due to nutritional disorders is not sufficiently realized. The author discusses the whole question of diet and lays stress upon the changes in our views regarding nutrition in recent times. Particular reference is made to the vitamins and their occurrence in various foods and absence in others is noted. The well-to-do classes tend to eat more than they require, while the labouring classes are habitually under-fed. Among the latter an ill-balanced diet is more common than one defective in all respects.

The Malay seldom undertakes strenuous labour and he eats rice which is merely husked without milling or steaming. Fish, game, eggs, fresh vegetables and fruits can be obtained and occasionally meat is eaten. Unless he changes his diet, therefore, the Malay seldom suffers from any food deficiency disease. As regards the Indian labourer, his diet consists of par-boiled rice with dal or peas invariably. Dried or fresh fish is occasionally consumed and meat rarely. Tapioca, sweet potatoes and green vegetables are often taken. Condiments containing essential oils are used in making curry and fat is supplied as cocoanut or gingeley oil. Fresh lime juice or tamarind pulp with salt are taken as savouries.

The diet of the Chinese labourer is supplied on a mass production scale. It consists of " polished rice of the lowest quality, salt fish, pork fat, sweet potatoes, tapioca and green vegetables either fresh or preserved. Preserved eggs and fish are occasionally included. No lime juice is employed. In its place, however, tow-yoo or stale vinegar is used. The vegetables and the pork fat or salt fish are mixed together and fried in kachang oil. These, with a little salt and vinegar, help him to eat his rice."

It appears, therefore, that the Malay and the Indian are fairly well supplied with vitamins B and C and to a less degree with vitamin A also. The diet of the Chinese, however, is lacking in vitamins B and C. The comparative frequency of scurvy, beriberi and pellagra among this race can thus be explained. In addition to the more definite food-deficiency diseases, other conditions are met with in the population

arising out of a diet too rich in carbohydrates and too poor in vitamins, proteins and inorganic salts. In infancy and childhood these take the form of rickets, dental caries, keratomalacia and catarrh of the respiratory and alimentary tracts. In adults similarly diarrhoea, dysentery, burning feet, night-blindness, trophic disorders of the skin, in addition to pellagra and beriberi, are encountered.

The author is of the opinion that " beriberi and pellagra are perhaps varied manifestations of a long process of intestinal intoxication induced by subsistence on a prolonged diet defective in vitamin B and protein with excess of starch and fat." It is evident from the above that much of the disease and ill-health in Malaya is preventable and it is urged that efforts should be made towards the local production of dietary articles of the right kind.

A. Douglas Bigland.

HEIN (Gordon E.) & MERRILL (John). **Pellagra.**—*California & Western Med.* 1929. May. Vol. 30. No. 5. pp. 334–338. [Med. School, Univ. of California, San Francisco.]

Both in the San Francisco Hospital and in the State of California pellagra has increased during recent years. Thus in 1920 one case was reported from the hospital and 16 in the State, while in 1925 there were 9 cases in hospital and 63 in the State. During the following year the figures were 13 and 58 respectively. Since 1924 the authors have had under observation 29 cases of the disease at the San Francisco Hospital. Of these 18 were men and 11 women. The disease usually attacked those over 45 years of age. A deficient diet was the most constant aetiological finding and unmarried men were more commonly affected. As regards race, it was noted that the disease occurred most frequently amongst the Irish. Seventeen of the men and seven of the women gave a history of excessive use of alcohol and it was thought that gastro-intestinal disease was also a predisposing factor.

On the clinical side there is little to note beyond the typical appearances. Out of eight gastric-juice examinations free HCl was absent in six. A mild secondary anaemia was the rule. Twelve of the 29 cases proved fatal. Of these, two died of tuberculosis without pellagra symptoms at the time and in ten alcoholic poisoning played a part. Autopsies were performed upon these cases dying of combined pellagra and alcoholism but nothing worthy of note was found. Short after-histories of some of the recovered cases are given.

A. D. B.

TURNER (Roy H.). **Pellagra associated with Organic Disease of the Gastro-Intestinal Tract.**—*Amer. Jl. Trop. Med.* 1929. Mar. Vol. 9. No. 2. pp. 129–137. [13 refs.] [School of Med., Tulane Univ. of Louisiana, & Charity Hosp., New Orleans.]

Pellagra associated with organic disease of the gastro-intestinal tract has been recorded fairly frequently. In 1916 ROLPH described a case with gastric carcinoma and since then BRYAN, BENDER and O'LEARY have recorded similar cases. JOYCE and SEABROOK have noted pellagra with rectal stricture, O'LEARY two cases with carcinoma of the descending colon, NUZUM two cases with carcinoma of the terminal ileum and ELLIOT one with carcinoma of the colon.

The material here presented was gathered from the wards and out-patient department of the Charity Hospital, New Orleans. Out of a total of 75 cases 16 showed pellagra associated with some organic disease of the alimentary tract. In only 15 cases was special search made for such disease and the author thinks that the number might have been increased if proctoscopic and X-ray examinations had been carried out more frequently. "The sixteen cases represent eight patients with stricture of the rectum, two with colitis—one probably tuberculous—and one of each of the following conditions: gumma of the stomach; recto-vaginal fistula and luetic proctitis; cloaca; partial stenosis of jejunum, due to old tuberculous ulcer; partial stenosis at the ileocaecal valve due to bands of adhesions, and amebic dysentery." The death rate was high, 50 per cent. dying in hospital in spite of thorough dietetic treatment. In the control group, without organic disease, the death rate was 25 per cent.

Gastric analyses were carried out in eleven cases. In spite of the severity of the disease free HCl was found in six. Of these eleven cases, eight had lesions in the colon or rectum. The three which had lesions in the stomach or small intestine all showed absent HCl. Only 38 per cent. of unselected pellagrins in another series showed the presence of free HCl. Though achlorhydria, in the author's opinion, is not an essential part of pellagra, it probably is an etiological factor of importance by reason of the increase in gastric and intestinal flora to which it gives rise. In this way harmful substances may be absorbed from the intestinal lumen.

During a period of nine months since this article was written the author states that ninety cases of pellagra were admitted to the Charity Hospital. Of these seven had stricture of the rectum and two had cancer of the stomach.

A. D. B.

GOLDBERGER (Joseph). **Pellagra.**—*Jl. Amer. Dietetic Assoc.* 1929. Mar. Vol. 4. No. 4. pp. 221–227. [Summary appears also in *Bulletin of Hygiene.*]

On a conservative estimate, 120,000 people in the United States suffered from an attack of pellagra during 1927. The disease is strikingly limited to the southern states and affects different states to a greatly different degree. Thus, while Virginia and Florida have been lightly affected, the Carolinas have been sorely stricken. There is a marked seasonal incidence, 80–90 per cent. of cases appearing in the period April–August, and there is a marked tendency to recurrence of the disease at this time. It is mainly a disease of children, though generally of a mild type, whilst the more severe cases are prone to occur amongst adult females. The endemic disease varies in its incidence with changing economic conditions, a decrease in purchasing power being liable to be followed by an increase in the incidence. It may now be stated that pellagra is a vitamin deficiency disease analogous to scurvy and beriberi. By omitting the pellagra-preventing (P–P) factor from the diet of dogs and rats a disease very similar to pellagra has been produced. The available evidence justifies the inference that all foods known to contain vitamin B contain the pellagra preventive. Foods rich in vitamin B are rich in the pellagra preventive, but the converse does not hold, for lean beef which is a poor source of vitamin B as judged by

the rat-growth test, is a very good source of P–P. The reasons for failure to include the P–P factor in the diet are various. Economic factors of all types play the greater part, but the sporadic cases occurring in well-to-do persons are due to finicky or faddy eating. The problem of eradicating the disease is complicated by dietary habits which are themselves, to some extent, related to climatic conditions and thus to the seasonal availability of certain foods. Improvement in basic economic conditions is the only general cure, though education of the people will help meanwhile.

H. N. H. Green.

DJAPARIDZE (P. S.). [La péllagre en Abhasie.] [**Pellagra in Abhasia.**]—*Russian Jl. Trop. Med.* 1928. Vol. 6. No. 10. pp. 624–634. With 2 text figs. [22 refs.] [In Russian. French summary p. 634.]

In July 1927 the author had occasion to investigate malaria and helminthiasis in Abhasia. In the course of this survey 22 cases of pellagra were noted. Of these, 10 were men, 12 women and 2 children. All belonged to the peasant class. Their diet contained unspoiled maize and often green vegetables and fruit, though two of the cases had never eaten maize at all. Treatment consisted of a five to six weeks' course of neo-salvarsan injections, followed by a longer course of arsenic given orally. The result was satisfactory.

[The actual total of cases mentioned was 22, but when sex incidence was investigated the number became 24, and when nationality was in question the number fell to 21. It is fortunate that the success of treatment did not depend upon simple mathematics ! !]

A. D. B.

ALSCHIBAJA (K.) [Pellagraepidemie in Mingrelien.] [**Epidemic of Pellagra in Mingrelia.**]—*Nachrichten der Tropischen Medizin.* Tiflis. 1929. Vol. 2. No. 5. pp. 332–335. With 2 text figs. [In Georgian script. German summary pp. 409–410.]

In the Georgian province of Mingrelia pellagra, which was formerly of rare occurrence, at the end of 1928 became epidemic. In the out-patient clinic at Sugdidi 107 cases were diagnosed. Of these 20 were men, 69 women and 18 children. All the classical symptoms were present.

Mingrelia is a country where much maize has been grown and consumed for centuries. In spite of this pellagra was rare. The author attributes this epidemic to a deficiency of good, unspoiled maize. Whereas formerly the people in good harvests were able to pick the best grains only, in recent years following several poor crops they were forced to import inferior maize and to harvest their fields before the crop was ripe.

A. D. B.

MACHWILADSE (Nico). Ueber die morphologischen Veränderungen des Blutes bei Pellagra. [**Morphological Blood Changes in Pellagra.**]—*Arch. f. Schiffs- u. Trop.-Hyg.* 1929. Mar. Vol. 33. No. 3. pp. 152–167. [32 refs.] [2nd Med. Clinic, State Univ., Tiflis, Georgia.]

The following blood changes are described in pellagra. The red cells are always reduced in number and this reduction bears no relation to the duration of the disease, but depends rather upon the severity of the case. The haemoglobin percentage is usually diminished and the colour index usually raised. The leucocytes are diminished in number, as are also the platelets, and the reduction of the latter is not propor-

tional to a similar reduction in other elements. The absolute lymphocyte count is usually normal, though the relative percentage is raised. An increase of the neutrophil percentage suggests complications, as in ordinary cases it is diminished. Basophil cells and monocytes are usually normal and any eosinophilia points to infection by parasites. The Arneth count shows a moderate shift to the left. Stained films show that the red cells vary very slightly from the normal.

From his observations the author concludes that pellagra is due to a toxin which acts upon the haemopoietic system.

A. D. B.

GAVRILA (I.). La vitesse de sédimentation des globules rouges dans la pellagre. [**Rate of Sedimentation of Red Cells in Pellagra.**]—*C.R. Soc. Biol.* 1929. Mar. 8. Vol. 100. No. 9. pp. 685–686.

The sedimentation rate of the red blood corpuscles has not previously been studied in pellagra. Observations are here recorded upon ten cases having characteristic skin lesions. The method of Westergreen-Katz was employed and in each case two examinations were made at two day intervals. Readings were taken half-hourly during two hours and again after twenty-four hours.

The results are shown in a table from which it is clear that the sedimentation rate is markedly increased in pellagra.

A. D. B.

CARRANCÁ (R.) & TRUJILLO. El maiz germinado en el tratamiento de la pelagra. [**Germinated Maize in the Treatment of Pellagra.**]—*Rev. Méd. Barcelona.* 1929. May. Year 6. Vol. 11. No. 65. pp. 439–441. [2 refs.]

The authors employ maize which has been kept in a moist atmosphere till it germinates, washed and triturated to a paste, for treating cases of pellagra. Water and sugar are added according to the patient's taste, the initial dose being 50–60 gm. daily. Sometimes mild symptoms of intolerance are evident in the earlier days—slight diarrhoea and a sense of fulness—but these soon pass off. They report very good results except in advanced stages.

H. Harold Scott.

GOLDBERGER (Joseph), WHEELER (G. A.), SYDENSTRICKER (Edgar), KING (Wilford I.) & others. **A Study of Endemic Pellagra in some Cotton-Mill Villages of South Carolina.**—*Hyg. Lab. Bull. No. 153.* Washington. 1929. Jan. pp. vi+85. With 20 figs. [26 refs.]

All the chief points of interest in this paper have already been noted in the *Bulletin* (*ante*, p. 356).

A. D. B.

## BERIBERI.

MITCHELL (J.). **Cases of Beri-Beri from the Persian Gulf.**—*Jl. Roy. Nav. Med. Serv.* 1929. Jan. Vol. 15. No. 1. pp. 44–47.

Eight cases of beriberi occurring in a ship serving in the Persian Gulf are described. The first case was noted in March, but it was thought that the ship's prolonged stay on the Trucial coast during May and June was largely responsible for the other cases. Three of the cases occurred at Bushire, where the disease is not endemic. From the case histories given it appears that cardiac symptoms predominated; in only two cases were the knee jerks absent. There were two deaths.

The first case was reported at a time when fresh and varied food was plentiful. Later fresh vegetables could not be obtained, the local meal was poor in quality, fish was scarce and the men were dependent on an insufficient weekly fruit ration from Bombay. Four of the cases were British ratings and the quantity of rice consumed by these was negligible.

In spite of the fact that tinned food formed a large part of the dietary during the summer, and that debility was prevalent among the men, the possibility of the infectious nature of the beriberi outbreak was entertained. The ship was therefore subjected to thorough weekly disinfection. Since it was impossible to obtain fresh vegetables the following arrangements were made, viz., dried peas were soaked for 48 hours previous to cooking, unpolished rice was substituted for the polished variety and yeast was given three times daily to all cases of debility.

A. Douglas Bigland.

JOURDRAN (E.) & LE MEILLOUR. Utilité des examens radioscopiques répétés au cours des affections cardio-vasculaires des béribériques. [**Radioscopic Examinations in Cardiovascular Beriberic States.**]—*Far Eastern Assoc. Trop. Med. Trans. Seventh Congress, British India, 1927.* Vol. 1. pp. 434–445. With 43 figs.

Alterations in the size of the heart in cases of beriberi observed radiologically by orthodiagrammatic methods are here described. In order to have an adequate series of controls three groups of the community were first carefully investigated by these methods. Series A comprised ten Annamites in hospital for surgical conditions without cardiac or pulmonary complications, series B was made up of ten hospital orderlies, and series C was chosen from among healthy prisoners.

The technique and the accurate measurements and diagrams figured and described would be of interest only to specialists. Suffice it to say that an increase in all the diameters of the heart cavities was found to be the rule in beriberi.

A. D. B.

MEBIUS (J.). Odemtheorie der Beri-Beri und physiologische Wirkung des Vitamins B. [**The Oedema Theory of Beriberi and the Physiological Action of Vitamin B.**]—*Virchows Arch. f. Path. Anat.* 1929. Feb. 27. Vol. 271. No. 2. pp. 432–449. [37 refs.] [Summary appears also in *Bulletin of Hygiene.*]

The author from careful examination of the hearts from three cases of acute beriberi observed that oedema of the cardiac muscle is present

and that this oedema is chiefly intracellular, affecting adversely the anisotropic substance of the muscle fibre. The excess of water in this situation naturally interferes with the normal fluid exchange within the fibre, and therefore with its contractility. This pathological condition in the heart muscle the author believes to be a primary phenomenon and not secondary to neuritis of the cardiac nerves. He goes on to show that the control of osmotic pressure depends upon the presence of vitamin B and that when this factor is deficient full muscular contractility is impossible owing to water absorption. Such a state of affairs obtains in beriberi and the disease is defined as one in which there is a disturbance in the osmotic pressure in the anisotropic substance of muscle.

The author then considers the question whether this alteration in the osmotic pressure can satisfactorily explain the whole picture of the disease. It is argued that the cardiac weakness, the low diastolic blood pressure, the muscular paralysis (including that of the diaphragm), the constipation, the anasarca and the paraesthesiae of beriberi are, in fact, due to this cause.

A. D. B.

MATSUMURA (S.), KAKINUMA (G.), KAWASHIMA (K.), TANIKAWA (K.), OCHIAI (S.), MIYATA (R.), FUJISAKI (K.), KANAO (R.), NOGUCHI (K.), AOKI (K.), SATO (T.), ITO (K.) & SUZUKI (M.). **The Etiology of Beriberi.**—*Jl. Amer. Med. Assoc.* 1929. Apr. 20. Vol. 92. No. 16. pp. 1325–1327. [Dept. of Hyg., Govt. Univ. of Med., Chiba, Japan.]

——. Estudos sobre a etiologia do kakkê ou beri-beri.—*Rev. Med.-Cirurg. do Brasil.* 1929. May. Vol. 37. No. 5. pp. 155–168. With 1 plate.

There are many facts concerning beriberi which the authors maintain cannot be explained by the vitamin hypothesis of the disease. Thus: (1) The main diet in Japan is rice all the year round. During the winter the fields are frozen and vegetables cannot be readily obtained. The people are therefore forced to subsist largely on rice, and yet beriberi occurs much more commonly in the summer. In sub-tropical and tropical areas such as Formosa, South China, French Indo-China, the Malay States and Java the disease prevails all the year round. (2) The incidence of beriberi appears to be associated with unsatisfactory environment. (3) When the disease occurs in a single situation, e.g., in a ship or factory, it tends to spread gradually, while others living on the same diet but in different surroundings may escape. (4). Overstrain, lack of exercise, insufficient sleep and intemperance predispose to beriberi. (5) Japanese statistics show that the epidemic tendency of beriberi varies from year to year just as it does in other epidemics.

Feeding experiments conducted by the authors show that pigeons fed on polished rice develop polyneuritis gallinarum in from two to seven weeks. This experimental disease can be divided into two types—the paralytic spasmodic form and the paralytic non-spasmodic form. The first responds well to vitamin B treatment, but if feeding with polished rice is continued, even when vitamin B is given also, a relapse occurs and the bird dies usually within nine days. In the second type even under the most favourable conditions " the injection of vitamin B is

a test of uncertain value." These results, in addition to the five points given above, are very difficult to explain by the vitamin deficiency hypothesis.

An organism resembling *B. coli communior* was found in relatively large numbers in the intestines of experimental animals and in human beriberi cases. The differentiation of this organism, which is called B. beriberi, from *B. coli communior* depends upon the fact that the former agglutinates and gives a complement-fixation reaction, while the latter does not. In a series of 32 pigeons fed upon polished rice two never developed B. beriberi in the faeces, while in the others periods varying from eight to 34 days elapsed before its appearance. Symptoms of disease were noted in from 4 to 14 days after the bacillus was found. Out of 135 cases of human beriberi the specific organism was present in the faeces of 98 (74 per cent.). The stools of non-beriberi cases were examined and here the incidence of the specific organism was only about 1 per cent. Fowls with beriberi show considerable agglutinative power, usually in titres of 200 to 500. Re-induction of the disease after initial recovery gave much higher figures, 1 : 1,000 or more. Taking 1 : 50 as the lowest titre of diagnostic value, in 30 consecutive human beriberi cases a positive result was obtained in 25 (83 per cent.), while in 40 normal persons the positive figure was only 7·5 per cent.

The authors claim that the beriberi bacillus " should be judged the principal aetiologic factor in experimental beriberi."

A. D. B.

TAYLOR (J.) & THANT (U.). **Observations on Vitamin B Deficiency in Pigeons (including the Occurrence of Refection).**—*Indian Jl. Med. Res.* 1929. Jan. Vol. 16. No. 3. pp. 747–765. With 5 charts. [5 refs.] [Pasteur Inst., Burma.] [Abridged from summary appearing in *Bulletin of Hygiene.*]

*Observations on beriberi columbarum.* Pigeons fed on certain natural items of Indian dietary deprived of the antineuritic fraction of vitamin B by the action of heat develop a condition termed " beriberi columbarum " by McCARRISON. In the present series of experiments the highest incidence of this condition occurred on a diet of autoclaved rice and dhal (a name given to various pulses), with a high protein content. That the condition was due to the deprivation of the antineuritic vitamin was shown by the fact that birds given unautoclaved dhal did not develop the condition. The post-mortem findings were in some cases masked by a starvation factor, but in other cases were typical and exactly the same changes were noticed in the heart, for instance, as are found in human beriberi.

*The effect of cooking on the antineuritic value of dhal and atta.* As stated above, dhal is the name given to various pulses, atta is a coarsely milled Indian wheat flour and is a staple food in northern India, where beriberi is rarely seen. It is little used in the southern parts of the country or in Burma, where the staple food is rice. Dhal is used to a certain extent by those whose staple dietary may be either rice or atta. It was found that in the cooked state dhal is not capable of furnishing a supply of vitamin B to replace adequately any deficiency in a rice dietary, atta being of the greatest value for this purpose. It is probable that the difference in vitamin B content of the atta and dhal may be due to the different methods of cooking employed.

A. F. Watson.

BANERJEE (Kali Gati). **On a Recent Outbreak of Epidemic Dropsy in the District of Birbhum.**—*Indian Med. Gaz.* 1929. Apr. Vol. 64. No. 4. pp. 181–182.

During the months October, November and December, 1928, the author saw 100 cases of " beriberi " in the district of Birbhum, Bengal. Clinically the majority had absent knee jerks, anaesthetic points over the shins occurred in some, all experienced great pain on squeezing the calf muscles and the squatting test was positive in only a few. Five types of the disease were noted : (1) *Febrile cases* : fever usually set in two or three days after the oedema of the legs appeared ; it did not rise above 101° and there was usually a fall in the evening. (2) *Dry cases* : here there was paresis of the legs and tender calves, but no oedema. Only one case of this type was seen. (3) *Gastro-intestinal type* : diarrhoea, indigestion, nausea and vomiting were present in about three-quarters of the cases. (4) *Fulminating or cardiac cases* associated with cardiac dilatation. One of these died on the seventh day. (5) *Simple cases* : these showed only oedema of the legs, most marked towards the evening, and they were able to carry on their ordinary duties. Further clinical details are given in a table comprising 28 cases.

The treatment was mostly dietetic. Patients and those residing in the affected area were instructed to take the following foods : " germinating grain ; oranges ; lemons ; tomatoes ; raw onion leaves ; cabbage leaves ; unboiled milk ; eggs—quarter boiled ; molasses ; yeast from the date palm (palmyra tree) ; bread ; khichuri, *i.e.*, a mixture of rice and whole grain mung or mossurie boiled together ; beans." Medicinally, various vitamin products together with general and cardiac tonics were administered.

In the author's opinion this epidemic was certainly due to the use of mustard oil. Whether the oil was infected with the spore-forming bacillus of *B. vulgatus* type (found in infected rice by ACTON and CHOPRA) was not determined [see this *Bulletin*, Vol. 22, p. 582]. Arguments are put forward against the infected rice theory of epidemic dropsy.

A. D. B.

GHOSH (U.). **Epidemic Dropsy in Birbhum.**—*Indian Med. Gaz.* 1929. Apr. Vol. 64. No. 4. pp. 182–183. With 1 map in text.

During November 1928 the author was called to see a household in Suri consisting of eleven persons, eight of whom had oedema of the feet. It was found that they all partook of mustard oil obtained from a single source in Sainthia about eleven miles away. Further cases were reported in other households in Suri and all had obtained mustard oil from the same source. Later it was found that 30 cases, confined to eight or nine families, had occurred in Sainthia itself and this was followed by outbreaks in the neighbouring villages. The distribution of the cases is shown by means of a map.

The outbreak was thought to be due to a particular consignment of mustard oil. It was discovered that the owner of the oil mill had received some four months previously four or five wagon loads of a particular seed, resembling pakra seeds. Also it appeared that a seed resembling mustard seed grows wild in the local jungles and this is

sometimes mixed with the real product. Such adulteration may possibly have been the cause of the outbreak. Neither vitamin B deficiency nor infected rice appeared to be responsible.

A. D. B.

DALAL (K. R.). **Report on the Investigation of an Outbreak of Epidemic Dropsy in Rangoon in the Year 1924.**—*Indian Med. Gaz.* 1929. Jan. Vol. 64. No. 1. pp. 19–20.

Particulars of an outbreak of epidemic dropsy, the first of its kind, occurring in Rangoon during the year 1924, are here supplied for the use of those interested in the epidemiological aspects of the disease.

Out of 174 cases recorded six proved fatal. Almost all the patients belonged to the Bengalee Hindu community. The outbreak was sudden in onset, the first cases being reported in July 1924. The maximum incidence was reached in the third week of July and by the middle of August the disease had practically disappeared. In most instances the various adult members of a family were attacked at very short intervals, but even in these families there was not a single case recorded among children living on milk diet only.

As regards the cause of the outbreak it is definitely asserted that neither vitamin nor other food deficiency was responsible, as most of the sufferers were well-to-do people. The impression gained at the time was that the probable causative factor was some infection or chemical toxin derived from a particular consignment of rice or mustard oil imported from Calcutta. Samples of both products were sent for analysis. It was found that most of the rice samples contained highly diseased grain, but the mustard oil gave negative results.

A. D. B.

PINTO (Wiseman). O beriberi é endémico em Góa ? (Comunicação). [**Is Beriberi Endemic in Goa ?**]—*Arquivos da Escola Med.-Cirurg. de Nova Goa.* 1929. Ser. B. No. 2. pp. 191–209.

Five cases of multiple neuritis accompanied by cardiac symptoms and some by oedema are briefly recorded, whose history, diet and clinical symptoms all coincide with true beriberi, and the author answers the question postulated in his title in the affirmative.

H. Harold Scott.

## HELMINTHIASIS.

PEVERELLI (P.). **Searching for Worm-Eggs with the Aid of Cedar-Oil Preparations.**—*Meded. Dienst d. Volksgezondheid in Nederl.-Indië.* 1928. Vol. 17. Pt. 4. pp. 569–572. [Med. Lab., Weltevreden.]

Peverelli has tested HEIN's method (*Jl. Lab. & Clin. Med.* Vol. 12, p. 1117) which consists in drying a thick smear of faeces at room temperature, putting on cedar oil and covering, thus rendering faecal matter transparent. The control was an " eosin preparation " and 105 samples of faeces were examined of which 7 were negative. The method is held to correspond to a thick blood film in malaria diagnosis and to offer the advantages of the concentration methods with the proviso that it "has not got their disadvantage that they often fail." Regarding results the eosin preparations failed in 24 instances in which the cedar oil preparations gave Trichuris 5, Ascaris 3, hookworms 6 [so presumably 24 should read 14] while the cedar oil failed 6 times, namely Trichuris 3, Ascaris 2 and hookworm 1. Both in the total number of eggs counted and in the time taken to find the first egg, the advantage of clearing with cedar oil [just as with glycerine] is marked with eggs whose shell is coloured, such being ascaris, trichuris and clonorchis. When ascaris loses its outer coloured shell this will not be so. For ankylostome eggs there is little difference between the results given by the two processes if such extremes as 50 minutes compared with 1½ minutes or 30 seconds with 30 minutes be excluded, and it is noted that these eggs may shrivel and disappear though not, it is held, if examination is made within half an hour.

Clayton Lane.

SMIRNOV (G.). [**Quantitative Helminthic Analysis. (Methods of Investigation).**]—*Rev. Microbiol., Epidémiol. et Parasit.* 1929. Vol. 8. No. 1. pp. 63–78. With 6 text figs. [Numerous refs.] [In Russian. English summary p. 118.]

The importance is emphasized of quantitative methods of diagnosis, that is of counts of worms and eggs, hookworm infection being in the writer's mind. Egg counts are preferred to worm counts and Clayton Lane's D.C.F. is declared the most accurate. Stoll's technique is held most acceptable for mass counting but owing to its ineffectiveness in light infections it must be controlled by D.C.F. Hung's method (the Hamburg cover glass) is the least accurate. Certain determination of the degree of infection from the number of eggs is clearly dependent on regular noncyclical oviposition, but in mass work such individual variations will be neutralized.

C. L.

GINESTET (Gustave). A propos de vers intestinaux. [**Intestinal Worms.**]—*Rev. Prat. Malad. des Pays Chauds.* 1929. Jan. Year 8. Vol. 9. No. 1. pp. 664–665.

In two cases of penetrating abdominal wound with exit in the buttock, portions of taenia escaped from the exit wound. In a third case a wound in the pubic region necessitated a median laparotomy ; a loop of small intestine disclosed a linear thickening about 20 cm. long with at one end a small patch where the intestinal wall was so thin that

perforation was imminent ; on incising this spot the thickening turned out to be an ascaris which had nearly perforated the intestinal wall.

C. L.

PIMENTEL (Candido). Tratamento moderno das helminthoses pelo tetrachloreto de carbonio. [**Treatment of Helminthiases by Carbon Tetrachloride.**]—*Folha Med.* 1929. July 5. Vol. 10. No. 19. pp. 224–225.

It is pointed out, without however mentioning the dosage used, that chenopodium has produced many cases of intoxication. It is held that absorption of carbon tetrachloride depends on its fluidity and that when put up in " solid solution " it is slowly absorbed. Accordingly Merck put it up in a solid which melted at body temperature. This melting being judged too rapid, the drug is now dispensed under the name " neonecatorina " in capsules in a matrix which does not completely melt till 50° C. is reached, each containing 0·7 gm. of the drug. The dosage of tetrachloride used, in years and grams is, 5 to 7, 0·7 gm. ; 8 to 10, 1·4 ; 11 to 13, 2·1 ; 14 to 16, 2·8 ; 17 and over 3·5. The method is held to be of incomparable value, eliminating in these doses all hookworms from laboratory animals of unstated weight, and as being efficient too against ascaris, taenia and strongyloides. Ten hookworms are stated to be more harmful than the equivalent weight of other parasites infesting man.

C. L.

FAUST (Ernest Carroll). **Parasiticidal Potencies of the Tri- to Hepta-Methyl Derivatives of Rosanilin.**—*Proc. Soc. Experim. Biol. & Med.* 1929. June. Vol. 26. No. 9. pp. 748–750. [3 refs.] [Peking Union Med. College, Peking, China, & Tulane Univ., New Orleans.]

In dilutions of 1 in 1,000,000 dahlia crystals (tri-methyl rosaniline) allow free growth of *B. subtilis*. The drug was without measurable effect against clonorchis in guineapig and pup. Methyl green (hepta-methyl para-rosaniline) was not measurably clonorchicidal, but was toxic to animals, and allowed free growth of *B. subtilis*. For this bacillus, gentian violet and methyl violet, which contain different mixtures of tetra-, penta-, and hexa-methyl para-rosaniline, were less toxic than crystal violet which is pure hexa-methyl para-rosaniline ; but as clonorchicides no difference between these compounds could be determined.

C. L.

LOSABERIDSE (E.). [Die Darmwurmerkrankung der Bevölkerung der Stadt Kutais und deren Kreises nach den Daten des kutaiser bakteriologischen Laboratoriums.] [**Intestinal Worm Infestation of the Town and District of Kutais, Georgia.**]—*Nachrichten der Tropischen Medizin.* Tiflis. 1929. Vol. 2. No. 4. pp. 299–302. [In Georgian script. German summary p. 326.]

From 1924 to 1927 this laboratory made 2,897 faecal examinations, presumably smears, 83·98 per cent. being positive for intestinal parasites. The percentage infection rates found were trichuris 42, ascaris 37 and ankylostomes 5 ; the last being very widely distributed. Protozoa were present in 3·9 per cent.

C. L.

SANDGROUND (J. H.). **A New Liver Fluke from a Monkey and New Parasitic Roundworms from Various African Animals.**—Reprinted from *Proc. U.S. Nat. Museum.* 1929. Vol. 75. Art. 12. pp. 1–11. With 9 figs. on 2 plates. [5 refs.]

The forms described are *Dicrocoelium colobusicola* n. sp. from the liver of a Colobus monkey ; *Leiperiatus* n. g. with *Nematodirus hopkeni* (Leiper, 1910) from the hippopotamus as its sole and therefore type species ; *Oswaldocruzia agamae* n. sp. from the intestine of *Agama colonorum* ; *Colobostrongylus strongi* n. g., n. sp., from *Colobus polykomos*.

C. L.

i. EL BATASH (M. H.). **The Nile as a Source of Infection with Schistosomiasis. A Survey of an Island in the Nile opposite Cairo.**—*Jl. Egyptian Med. Assoc.* 1929. Apr. Vol. 12. No. 4. pp. 41–46.

ii. HELMY (M. M.). **Observations on the Presence in the Nile in Egypt of the Molluscan Intermediate Host of** *Schistosoma haematobium.*—*Ibid.* pp. 47–49. With 3 figs. on 1 plate. [4 refs. ]

iii. ONSY BEY (Anis). **Bilharzial Infection of the Spleen and Egyptian Splenomegaly.**—*Ibid.* pp. 50–53. With 2 figs.

iv. FOUAD (Abdel Megid Mohammed). **A New Method for detecting** *Schistosoma mansoni* **Ova in the Stools.**—*Ibid.* p. 54.

i. In Rodah Island opposite Cairo examination, by unstated techniques, gave percentages of infection as follows, *S. haematobium* 25·5, ankylostomes 18, ascaris 3·7, *H. nana* 17·6, trichostrongylus 1. The snails found on the shores are listed. Planorbis and *S. mansoni* were absent.

ii. A water plant, *Potamogeton crispus,* appears particularly pleasing to Bullinus, and is broadcast in large masses into river and canals, with adherent snails and their eggs, during the Nile flood.

iii. It is now believed that bilharzia is an important factor in Egyptian splenomegaly, the findings being nodules accompanied by active multinucleated giant cells digesting the schistosome eggs. There follow eosinophilia and fibrosis.

iv. The deposit from concentrated salt solution is pipetted off and examined.

C. L.

GOLDIE (Horatio). Zur Pathologie und Therapie der chronischen Bilharziosis. [**Pathology and Treatment of Chronic Schistosomiasis.**]—*Arch. f. Schiffs- u. Trop. Hyg.* 1929. Apr. Vol. 33. No. 4. pp. 198–210. [22 refs.] [" Hadassa " Polyclinic, Tiberias, Palestine.]

*Schistosoma haematobium* is not endemic about Tiberias nor has Bullinus been found there. Goldie has, however, met there two classes of urinary schistosomiasis, established by finding the eggs. In the one, onset is acute, there has been bathing in pools in Egypt or in streams in Nahr el Auja by men of 17 to 32, the symptoms are urinary and indeed in some cases the men had been saddled with a diagnosis of gonorrhoea and treated for it ; these cases cleared up with tartar emetic, the

criterion of cure being absence of ova a month after cessation of treatment. The other class was quite different. When Goldie instituted routine microscopic examination of the urine, schistosome eggs were found in 28 Kurdish Jews, nearly all women, who came up for anaemia, menstrual trouble, lumbar or hypogastric pain, and pneumonia, acute or chronic, from Arwil, Erbil or Arbail, the ancient Arbela in Iraq. Cellular elements in the urine were generally few or none, and the condition had lasted generally from 3 to 5 and in one case 10 years. Tartar emetic proved an unsatisfactory remedy. Antimosan (40 to 75 cc. in all) was used in 19 cases, 14 being negative for eggs 3 months later.

C. L.

KHALIL (M.), NAZMI (M.), PETER (F. M.), EL DIN (M. Salah) & EL BETASH (M. H.). Die Behandlung der Schistosomiasis mit intramuskulären "Fuadin"-Injektionen. [**Treatment of Schistosomiasis with Intramuscular "Fuadin" Injections.**]—*Deut. Med. Woch.* 1929. July 5. Vol. 55. No. 27. pp. 1125–1126. [2 refs.] [Faculty of Med., Cairo.]

Fuadin, an antimonial preparation propitious for Egyptian use, was given intramuscularly to 300 schistosomiasis cases in 7 per cent. solution. The adult dosage was in the last 50 cases 1·5 cc. on the first day, 3·5 cc. on the second and 5 cc. on the third. Thereafter the last dosage was continued on alternate days up to the fifteenth, or nine injections in all. No death occurred. Comparing this experience with that of tartar emetic, of which more than 400,000 intravenous injections were given in Egypt during 1928, it is concluded that fuadin gives slight or no local reaction as compared with occasional inflammation, abscess or necrosis with tartar emetic; no general reaction as compared with frequent cough, vomiting and fainting; late vomiting in 2·5 per cent. of cases compared with fever, continued vomiting, jaundice and sudden death with tartar emetic, the statement being unqualified; a two-weeks instead of a four-weeks treatment; but at a relatively high instead of a low cost. If living eggs are found after the ninth injection one or two more must be given; yet sometimes the lethal effect on eggs is observable only after the course has stopped, indicating a cumulative effect. A two-way syringe allows of the use of a common solution, the needles being sterilized for each case.

C. L.

OZAWA (Makoto). [**Clinical, Histological, and Statistical Observations on *Schistosomiasis japonica* as the Cause of Acute Appendicitis.**]—*Jikken Igaku Zasshi.* 1928. Nov. Vol. 7. No. 1. [Summarized in *Japan Med. World.* 1929. Mar. 15. Vol. 9. No. 3. p. 93.]

Ozawa has compared the histological findings of 20 cases of appendicitis (4 acute and 16 chronic) with those of six cases of other surgical intestinal affections, infection with *Schistosoma japonicum* being common to all. Eggs were present in all cases, but inflammatory changes in the former only, and with them bacterial infections. Moreover, it is stated that many cases of appendicitis occur in localities where schistosome infection is endemic and very few where it is not. Accordingly he concludes that there is a close relationship between the schistosome and appendicitis.

C. L.

i. HARRIS, Jr. (H. Elwin). **Appendicitis caused by Bilharzia.**—*Brit. Med. Jl.* 1929. May 4. pp. 810–811.

ii. PERRY (H. Marrian) & BENSTED (Harold J.). **Appendicitis as a Complication of Bilharzial Infection.** [Correspondence.]—*Ibid.* June 8. pp. 1059–1060.

i. The record is of a native of the Nile Delta whose appendix was removed after his third attack of appendicitis. It was bright red and contained ova of *Schistosoma haematobium*, some calcified, with surrounding engorged vessels and extravasated leucocytes. The blood showed a 12 per cent. eosinophilia. "I have been unable to find any instance of appendicitis caused by this parasite."

ii. In Egypt over 50 per cent. of appendices removed for appendicitis show the picture described above. "It is questionable, however, whether the inflammatory process is due in all cases to the presence of these sources of irritation."

C. L.

i. JAGUBOFF (F.). [Trematodosen der Leber bei Menschen in Aserbeidschan.] [**Liver Trematodes in Men of Aserbaijan.**]—*Rev. Microbiol., Epidémiol. et Parasit.* 1929. Vol. 8. No. 1. pp. 40–44. [5 refs.] [In Russian. German summary pp. 114–115.] [State Inst. for Microbiol. & Hyg., Baku.]

ii. PLAVTOV (K.). [**Distomiases among the Population of Nakhichevan.**]—*Ibid.* pp. 49–53. [In Russian. English summary pp. 116–117.] [People's Commissariat for Health, Nakhichevan.]

iii. KARIBOW (N.). [Zwei Fälle der *Fasciola hepatica* Inwasion.] [**Two Cases of** *F. hepatica* **Infection.**]—*Nachrichten d. Tropischen Medizin.* Tiflis. 1928. Nov. Vol. 1. No. 1. [In Georgian script. German summary p. 113.]

iv. KOULAGUINE (S. M.). Transit d'oeufs de trématodes par le tractus intestinal de l'homme.] [**Passage of Trematode Eggs by the Intestinal Tract of Man.**]—*Russian Jl. Trop. Med.* 1929. Vol 7. No. 2. pp. 131–135. [In Russian. French summary pp. 135–136.]

i. In the course of examining 900 men during 4 months, trematode eggs were found in 3. In the first *Fasciola hepatica* eggs were detected in 13 of 14 examinations spread over two-and-a-half months, the liver being greatly enlarged. In the second the same eggs were found in a similar case. In the third *Dicrocoelium lanceatum* ova were seen on each of 3 examinations; the liver being greatly enlarged and tender, haemoglobin 18 to 25, red corpuscles 1,600,000 to 1,800,000. He was also infected with hookworms. In a note Professor SDRODOWSKI adds another and similar case of infection with *D. lanceatum*

ii. By unstated methods the following infections were found, apparently in 909 persons—Ascaris 444, Trichuris 343, *Taenia saginata* 128, *Hymenolepis nana* 30, Oxyuris 20, Trichostrongylus 8, *Dicrocoelium lanceatum* 8, *Fasciola hepatica* 1. Local sheep were heavily infected with the last two and Limnaeus and Planorbis exist. A case of *D. lanceatum* infection, apparently typical of the eight, had loose stools, vomiting, weakness and anaemia, that is symptoms not necessarily implicating the liver.

iii. Two boys of 11 in different Georgian provinces suffered from anaemia and eosinophilia. One had biliary colic, the other had not. Fasciola eggs were found in the duodenum by sound and in the faeces. Emetine caused eggs and symptoms to disappear.

iv. Struck by finding eggs of *Fasciola hepatica* and *Dicrocoelium lanceatum* in 40 persons, two only of whom showed liver symptoms (one of each infection), Koulaguine suspected that the eggs passed in the faeces might have been taken in as such with food. He was able to observe a series of cases in which ingestion of boiled infected liver resulted in the passage of eggs of these two species. Having by faecal examination satisfied himself that his own faeces were free from eggs, he took a pâté of infected liver. Eggs of both species appeared in the faeces next day and persisted for 3 days. Swallowed eggs are freely recognized after ordinary cooking. It is concluded that the presence of eggs should not be taken to signify infection, unless eating of infected liver can be excluded, the subject is observed over a period, or eggs have been recovered by duodenal catheter.

C. L.

STUNKARD (H. W.) & WILLEY (Chas. H.). **The Development of Cryptocotyle (Heterophyidae) in its Final Host.**—*Amer. Jl. Trop. Med.* 1929. Mar. Vol. 9. No. 2. pp. 117–128. With 6 figs. [15 refs.] [Biol. Lab., New York Univ., New York.]

The authors' conclusions are as follows :—

"A review is presented of the species of the Heterophyidae known to infest man. They have been placed in the four genera, Heterophyes, Metagonimus, Stamnosoma, and Monorchotrema. The two species *Monorchotrema taihokui* and *M. taichui* are compared and shown to be identical. Experimental study of the life history of the heterophyid trematodes shows that they develop in various birds and mammals, including the common laboratory animals, and the suggestion is made that all are probably human parasites. Study of the reports of Yokogawa, Ciurea, and Faust and Nishigori, shows that their conclusion (that the metacercariae of Metagonimus, Apophallus, and Monorchotrema, respectively, when liberated from their cysts bore into the intestinal wall and develop there to sexual maturity) is not well substantiated. Experiments conducted with laboratory animals demonstrate that Cryptocotyle does not have such an intramucosal stage in its life history."

Reproduced microphotographs show the young worms between the villi.

C. L.

WITENBERG (G.). **Studies on the Trematode—Family** *Heterophyidae.*—*Ann. Trop. Med. & Parasit.* 1929. June 27. Vol. 23. No. 2. pp. 131–239. With 33 text figs. [6 pages of refs.] [Dept. of Parasit., Hebrew Univ., Jerusalem.]

This considerable paper defines the Heterophyidae, subdivides them into sub-families on the bodily shape, circumoral spines and number and position of testes, and into genera on the arrangements of the genital glands and various structures of the ventro-genital sac which includes the genital sucker or "gonotyl." Three species of *Heterophyes* are accepted, *H. aequalis*, *H. dispar* and *H. heterophyes*: *N. nocens* is retained provisionally. *Metagonimus romanicus* and *M. dobrogiensis* are held identical with one another and probably with *M. yokogawai*.

C. L.

JOYEUX (Ch.) & BAER (J. G.). Les cestodes rares de l'homme. [**Rare Cestodes of Man.**]—*Bull. Soc. Path. Exot.* 1929. Feb. 13. Vol. 22. No. 2. pp. 114–136. [57 refs.]

A critical consideration of the cestodes of man particularly those rarely reported. *Diphyllobothrium latum* has as synonyms *D. cristatum, D. taenioides, D. parvum* and *D. minus*; while *D. cordatum* and *Diplogonoporus grandis* are strays. *Braunia jassyensis* the authors have had the advantage of examining. The specimen is immature, held to be a larval ligula and the creation of a genus for it premature. *Digramma brauni* is based on immature segments and is probably a known bird species too immature to identify. The species of *Bertiella* were reduced by BAER (1927) to two, *B. studeri* of the Old World and *B. mucronata* of the New. *B. satyri* is a synonym of the former. Of the five genera into which *Davainea* has been divided *Raillietina* alone infects man. *Raillietina* (*Raillietina*) *formosana* is held to be a synonym of *R. celebensis*. In *R.* (*R.*) *madagascariensis* more than one species is concerned, but facts are insufficient to permit of their differentiation; *R. demerariensis* differs from these, but the immature segments they have examined permit of no recognition of the subgenus to which it belongs. On its single record "*Davainea asiatica*" cannot be placed. As synonyms of *Taenia saginata* come *T. confusa, T. africana, T. hominis, T. tonkinensis, T. philippina, T. bremneri*, and *T. cylindrica*. *Taenia infantis* is really *T. taeniaeformis*.

C. L.

KARGER (P.). Bandwurmkuren bei Kindern. [**Treatment for Tape Worm in Children.**]—*Med. Klin.* 1929. May 10. Vol. 25. No. 19 (1274). pp. 745–746. [1 ref.] [Children's Clinic, Univ., Berlin.]

Many tapeworms are treated in this Clinic. Primary stress is laid on the purge. In each case trials are made in order to discover one which the child will take readily and which will produce copious thin evacuations. Generally this can be effected by a teaspoonful of "Kurellaschem Brustpulver." The anthelmintic is extractum filicis and the dose for older children 8 gm., or even 10 gm., given fasting in the morning in two portions at half an hour's interval, followed in an hour by the selected laxative. If there has been no vomiting this treatment has been without failure. In case of vomiting a duodenal sound is passed through the nose. This in no way disturbs the child and X-rays have shown that 4 hours later its point is always in the duodenum. The medicament is then run in and comes in contact with the head of the worm in concentrated form, for H. SCHNEIDER has actually brought away a tapeworm head in the eye of such a sound. This treatment, it is stated, is not as complicated as it seems.

C. L.

BARRON (Moses). **Infestation with** *Diphyllobothrium latum*, **Fish Tapeworm: with Especial Reference to Native Cases.**—*Jl. Amer. Med. Assoc.* 1929. May 11. Vol. 92. No. 19. pp. 1587–1593. With 8 text figs. [23 refs.]

Barron adds 3 to a list of 16 cases of *D. latum* infection in America-born persons, and deals also with 19 cases of this infection which have

come under his personal observation. The subjects had lived in America 4 to 36 years. In those tested, eosinophilia varied from 0·5 to 12. In only one case was there no history of tasting raw fish, the customary procedure when preparing the Jewish delicacy of "gefüllte" fish, seasoned before cooking. In his cases there were no characteristic symptoms. He points out that in Finland "pernicious" anaemia occurs in about 1 in 6,000 of carriers of the broad tapeworm. Barron reports one case of "pernicious" anaemia in which infestation was discovered on the second admission to hospital only, nor did disinfestation cure the anaemia—she died of it. Treatment is with 6 gm. of oleoresin of aspidium, the male fern, with fasting and purging. The head may be missing without recurrence.

C. L.

FAUST (Ernest Carroll). **What is** *Sparganum mansoni?—Jl. Trop. Med. & Hyg.* 1929. Mar. 15. Vol. 32. No. 6. pp. 76–77. [Dept. of Trop. Med., Tulane Univ., New Orleans.]

Faust's reply to the question in his title is that "*Sparganum mansoni*" has been used for all unbranched spargana recovered from man and that several species have identical shape. They can be distinguished only by producing development to the adult. From the point of view of nomenclature, since the original form studied by COBBOLD is unrecognizable and since JOYEUX (1927) is the first worker to have used the species name "*mansoni*" in a recognizable description of an adult worm developed experimentally from the sparganum stage, the original term (*Sparganum mansoni* Cobbold, 1883) must give way to the amended or restricted one of Joyeux, by which is meant the sparganum stage of "*Diphyllobothrium mansoni* Joyeux, 1927." The normal intermediate host of this worm is *Rana esculenta* and A. S. CAMPBELL of Foochow, China has obtained, from two patients from whose fingers he extracted spargana, histories that freshly split frogs had been applied to them as poultices.

C. L.

JOYEUX (Ch.) & BAER (J. G.). Recherches sur le cycle évolutif d'*Hymenolepis fraterna* Stiles. [**The Developmental Cycle of** *H. fraterna.*]—*C.R. Soc. Biol.* 1928. Nov. 2. Vol. 99. No. 30. pp. 1317–1318.

BACIGALUPO of Buenos Aires has made unpublished researches which lead the authors to conclude that his *Hymenolepis intermedius* (which their *H. bacigalupoi* replaced owing to preoccupation) is identical with *H. fraterna.* In Paris the authors have succeeded in transmitting *H. fraterna* without intermediate host in 100 per cent. of their experiments. Failure only occurred when the rats used were old or already spontaneously infected. *H. fraterna* was never found by the authors in Tenebrio or other insect, although BACIGALUPO has found naturally infected Tenebrio in Brazil.

R. T. Leiper.

LARROUSSE (F.). Hôtes intermédiaires nouveaux d'un cestode de la souris *Hymenolepis microstoma* (Dujardin, 1845). [**New Intermediate Hosts of** *H. microstoma.*]—*C. R. Soc. Biol.* 1929. Apr. 8. Vol. 100. No. 11. pp. 855–857. With 1 text fig. [2 refs.] [Inst. of Hyg. & Bact., Strasbourg.]

Cysticercoids identified by JOYEUX as *Hymenolepis microstoma* Dujardin were found by Larrousse in *Gnathocerus cornutus* Fabr., and *Trogosita mauritanica.* The former insect is illustrated. Both are Coleoptera.

C. L.

BACIGALUPO (Juan). Un nuevo huésped intermediario del *Hymenolepis diminuta* (Rudolphi 1819), el *Ulosonia parvicornis* Fairmaire 1892. [**A New Intermediary Host of** *H. diminuta.*]—*Semana Méd.* 1929. May 23. Vol. 36. No. 21 (1845). pp. 1339–1340. With 2 text figs.

Two of three specimens of *Ulosonia parvicornis* collected from rat hutches contained in their body cavities cercocyts of *Hymenolepis diminuta.* Of 14, collected from hutches containing guineapigs and rabbits, two were free of infection. The other 12 were fed on eggs of the tapeworm; the results are to be given later. Larval stages of this small coleopteron were uninfected.

C. L.

RANIERI (Gustavo). Sindromi rare di parassitosi intestinale. Nota I. Teniasi a sindrome appendicolare e pseudoneoplastica. [**Taenia Infestation with Appendicular Symptoms.**]—*Riforma Med.* 1929. Feb. 23. Vol. 45. No. 8. pp. 255–256. With 2 text figs.

A man of 37 suffered from symptoms suggesting appendicular colic, looked well, had no fever, but had a visible and palpable tender elongated swelling in the ileo-caecal region. X-rays after opaque meal and enema showed a clear area in this region, tapeworm eggs were present in the faeces, an anthelmintic expelled *Taenia saginata,* and recovery followed.

C. L.

ABULADSE (Sol). [Zu der Methodik der Anfertigung der Dauerpräparate von Bandwurmproglotiden.] [**Permanent Preparations of Tapeworm Proglottides.**]—*Nachrichten d. Tropischen Medizin.* Tiflis. 1928. Dec. Vol. 1. No. 2. [In Georgian script. German summary p. 226.]

The washed flattened preparation is placed in Hofer's solution for 24 hours, washed for a few minutes in 50 per cent., and left for 2 to 4 days in 70 per cent., alcohol. It is then removed from between the slides which have kept it flattened and allowed to dry for some days. Picric acid crystallizes out in the uterine cavity and displays this from the genital opening to all branchings. The dried proglottis is then mounted in Canada balsam.

C. L.

FAIRLEY (Keith D.). **The Intradermal Test in Hydatid Disease: a Critical Analysis of its Results.**—*Med. Jl. Australia.* 1929. Apr. 13. 16th Year. Vol. 1. No. 15. pp. 472–483. [30 refs.] [Walter & Eliza Hall Inst. of Research, Melbourne.]

The technique was the injection intradermally of 0·3 to 0·4 cc. of pooled aseptic active hydatid fluid the needle being bevel upwards; a

few centimetres away a control injection of 0·85 per cent. saline was made. The wheal caused by an injection forms a white raised area like pigskin about 1 cm. in diameter. The control rapidly fades. A positive reaction is immediate or deferred. When immediate the wheal increases in size usually with irregular processes and is surrounded by erythema ; the maximum is reached in 20 minutes ; it has faded in an hour. The delayed reaction appears locally usually some hours later as an indurated hot usually red swelling with a very definite edge ; it has usually disappeared in 36 to 72 hours but may persist a week and may cover the whole limb but without glandular enlargement. Preoperative reactions in 35 persons with uncomplicated hydatids gave an immediate reaction in 77 per cent. and a delayed one in 56. When cysts had ruptured or were suppurating these percentages were 92·6 and 26 in 27 cases. Of 87 persons showing immediate reaction there was proof of hydatid disease in 59·8 per cent., so that no one should be operated upon on this finding alone. When there is no history of recent urticaria, absence of immediate reaction definitely excludes a ruptured or suppurating cyst. Absence of a delayed reaction is valueless in excluding hydatid, but if associated with an immediate response, and a high titre complement fixation or precipitin test, is frequent in the second week after operation, and in the absence of an operation implies rupture or suppuration. Reactions persisting ten years or more after operation demand careful scrutiny for further cysts.

C. L.

KELLAWAY (C. H.). **Anaphylactic Studies with Extracts of Hydatid Scolices.**—*Brit. Jl. Experim. Path.* 1929. Apr. Vol. 10. No. 2. pp. 115–125. With 3 text figs. [11 refs.] [Walter & Eliza Hall Inst. of Research, Melbourne.]

Summary :—

" The general result of these experiments is to bring the hydatid scolex into line with other helminths, the tape-worm (Meyer) and the fluke (Kellaway). The substances in scolex which are insoluble in acetone but soluble in absolute alcohol and which act as ' partial antigens,' causing sensitiveness but being unable to discharge it, may possibly owe their activity *in vivo* to union with some body protein in the guineapig producing a foreign complex to which antibody can be produced. There is, however, in addition a water-soluble substance present in saline extracts of scolices which can function as an anaphylactic antigen. Owing to the difficulty of freeing scolices from the protein substances in hydatid fluid it is not certain that this substance is not derived from hydatid fluid. Finally host (sheep) serum protein is present in extracts of scolices and extraction with pure dry acetone and with absolute alcohol does not guarantee its absence from the resulting extracts."

C. L.

CARMALT-JONES (D. W.). **Hydatid Disease as a Clinical Problem: Some New Zealand Experiences.**—*Brit. Med. Jl.* 1929. July 6. pp. 5–9. [10 refs.]

Although in New Zealand when dealing with obscure symptoms or signs the injunction is " think of hydatid," the total cases number only some 150 a year of which 7 or 8 come yearly to the Dunedin hospital and are distributed among as many medical men. Since deaths in non-suppurating cases were 6·3 per cent. and in suppurating cysts

52·7 per cent. the importance of anticipating suppuration by operation is stressed. Recently devised tests, such as the Casoni skin test, complement fixation and improved skiagraphy have raised the rate of correct diagnosis from 40 to 80 per cent. Illustrative cases show how the patient's or the family history and the Casoni test may help, and yet how the laboratory tests may mislead and extraction of fluid clinch the diagnosis.

C. L.

COUTELEN (F.). Action *in vitro* des radiations ultra-violettes sur le sable hydatique. [**Action of U.V. Rays in Vitro on Hydatid Sand.**]—*Ann. Parasit. Humaine et Comparée.* 1929. July 1. Vol. 7. No. 4. pp. 274–279. [18 refs.]

With ultraviolet rays of an intensity which will kill staphylococci in 5 to 10 seconds, the *coli* group in 18 to 19 seconds, *subtilis* in 45 seconds and the tubercle bacillus in a minute, hydatid scolices show for an hour excited movements and are not all dead until after 2½ hours' irradiation.

C. L.

FAIRLEY (N. Hamilton) & WRIGHT-SMITH (R. J.). **Hydatid Infestation** (*Echinococcus granulosus*) **in Sheep, Oxen and Pigs, with Special Reference to Daughter Cyst Formation.**—*Jl. Path. & Bact.* 1929. Apr. Vol. 32. No. 2. pp. 309–335. With 21 figs. on 11 plates (1 coloured). [29 refs.] [Walter & Eliza Hall Research Inst., Melbourne.]

This investigation deals with 11,257 sheep (16·5 per cent. infected), 4,922 cattle (23·9 per cent. infected), and 2,497 pigs (0·5 per cent. infected).

The sheep constitutes the hygienically important intermediate host in Australia, because of the high incidence of infection, the high percentage of fertility associated with high intracystic tension and a thick membrane, and the frequency with which infected offal is fed to dogs. The high tension within the actively growing cyst causes it to enlarge irregularly where resistance is least (between such obstructions to expansion as vessels, bile ducts and bronchii) so producing the multilocular type. The ratio of lung to liver site was 1·13 to 1, and of right to left lung 1·36 to 1. In 17, cysts were too numerous to count, and in 14 so widespread as to suggest a blood-borne infestation. Degeneration was found in 13·57 per cent. of 6,497 cysts, and was higher in lung than liver ; daughter cysts were met with in 146 of these, 117 exogenously and 29 endogenously. Of the former 70 per cent. were healthy ; of the latter all were absolutely so with a fertile mother cyst, while with one partial exception the adventitia was equally normal.

In connexion with the suggestion based on observation in man that endogenous daughter cyst formation is defensive against inimical forces, the authors urge that only in short-lived hosts can the true early state of the mother cyst be observed ; their observations in sheep, ox and pig " conclusively show that in the food-animals under review mother cysts containing single or multiple endogenous daughter cysts are intact. fertile; perfectly healthy, contain fluid under normal tension and present stigmata of degeneration neither in the maternal cyst wall nor in the adventitia." The condition is the reverse ; degeneration in old mother cysts is secondary to increased cystic tension the result of growth of secretion of young endogenous daughter cysts.

In the ox, although 23·9 per cent. are infected, by far the commonest infection is with simple sterile unilocular cysts nearly always less than 2 in. in diameter; other types in order of frequency are unilocular fertile, multilocular and multicystic. Multilocular cysts are fortunately much less common; for if daughter cysts occur (they are nearly always exogenous) they have a high brood capsule content. The multicystic condition (the *Echinococcus multilocularis* of veterinarians) is regarded as caused by progressive early exogenous budding in the liver (it does not occur in the lung) before an adventitial barrier has formed. Degeneration is frequent in hepatic bovine cysts, but was found in in 16 per cent. only of lung cysts, although 90·4 per cent. of lung cysts were sterile. Exogenous cysts, specially considered in the ox, begin as nipple-like external projections into the pericystic space in one localized area. As they grow they separate the healthy mother cyst from the adventitia and indent the latter. Increasing tension leads to death and degeneration of either mother or daughters, usually of the mother cyst which is collapsed, central, degenerate and often folded.

In pig the multicystic form was absent and the multilocular rare. A distinctive feature is hyaline degeneration of the cyst membrane showing itself either as a blancmange-like mould of the whole cavity or as clusters of pseudo-daughter cysts within the laminated membrane and often bulging inwards like daughter cysts. "It was not until detailed microscopic study . . . had revealed the constant absence of germinal membrane, hydatid elements, or indeed nuclear structures of any kind in the walls of these cysts that final proof of their essential nature was forthcoming . . . In man we have observed them in all stages including single isolated cysts, clusters, and finally honeycombing involving the whole thickness of the laminated layer." Failure to find other types of cyst with intracuticular origin leads the writers to the conclusion that "daughter cysts" so described are really due to hyaline cystic degeneration.

Regarding endogenous daughter cysts the only valid evidence is regarded as indicating their origin from living scolices and the authors support DÉVÉ in the conclusion that their most common source is vesiculation of a mature scolex.

C. L.

DEW (Harold). **Secondary Echinococcosis.**—*Jl. College of Surgeons Australasia.* 1929. Mar. Vol. 1. No. 3. pp. 337–350. With 8 text figs. [4 refs.]

This paper's essence lies in the following sentence: "From these observations, therefore, emerges the fact that daughter cysts, brood capsules, scolices and germinal membrane can all, if shed into the tissues under aseptic conditions, implant and give rise to secondary cysts that may ultimately become fertile." The shedding may occur either into a visceral cavity—peritoneum, pleura or pericardium; or into the circulation, venous comprising a peripheral vein or the right side of the heart, or arterial comprising the left chambers of the heart. The clinical picture of a cardiac hydatid shows typically a primary cyst growing for 5 to 10 years, its anaphylactic rupture, 2 to 5 years' latency, signs of cysts in pericardium, lung or brain; and lastly "a second intracardiac rupture of the now multivesicular original

cyst with as a rule anaphylactic shock and death from daughter cyst embolism of the cerebral vessels." Dew writes of the effect of injection of washed viable scolices into rabbits :—

"The vesicular change in the scolex is a relatively simple process. The scolex enlarges, becomes hydropic and the cells become spread out on the inner aspect of the cuticle which in turn rapidly thickens and becomes laminated. The only structures that persist, are the hooklets which are found aggregated in a confused mass at one pole, but as the cyst grows, they too become scattered and disappear. Striking features of the experiments which are also borne out by many clinical observations, are the facts that although a great number of scolices may be set free in the tissues, the resultant cysts are never very numerous, probably rarely more than 5 per cent. of the number of scolices and that of those that do survive, many undergo degenerative changes comparatively early. There is no doubt that many are overwhelmed by the cellular reaction of the host and undergo phagocytosis and fibrosis."

C. L.

HASEGAWA (K.). Ueber die Widerstandsfähigkeit der reifen Larven der parasitären Nematoden. [**Capacity for Resistance of Mature Larvae of Parasitic Nematodes.**]—*Taiwan Igakkai Zasshi* (*Jl. Med. Assoc. Formosa*). 1929. June. No. 291. pp. 27–28. [Med. School, Taihoku.]

The author repeats experiments often made, dealing with the actiou of chemical substances on larvae of hookworms, strongyloides and trichostrongylus. He claims however, that larvae kept in 4 to 6 per cent. salt solution for some days will yet revive in water, but will not do so if left in the salt for a month or two. It is held, too, that larvae of Ancylostoma revive after 87 hours' drying at 27° C. to 32° C., *Trichostrongylus orientalis* after 53 days and *Strongyloides stercoralis* after 18 minutes.

C. L.

LANE (Clayton); MANSON-BAHR (Philip). **The Mechanism of Filarial Periodicity.**—*Lancet.* 1929. June 22. pp. 1291–1293. With 1 text fig. [12 refs.]; July 6. pp. 45–47. [2 refs.]

In this stimulating and important paper the vexed question of filarial periodicity with special reference to *Filaria bancrofti* is dealt with. The old Mansonian view of a constant micro-filarial population sheltered during the day in the lungs and internal vessels and reaching the cutaneous capillaries only at night is rejected mainly on the grounds that no secret retreat in the viscera has been demonstrated during life, while the embryos which have no means of fixation in the large blood vessels, accumulate there only at or after death. The mechanical possibility of microfilariae being held back in the visceral capillaries is admitted but it is argued that the capillary bed could hardly be closed for one species (*F. loa*) and simultaneously in the same host be opened for another (*F. bancrofti* in India). An alternative hypothesis regarding periodicity is formulated. In it the author postulates simultaneous cyclical parturition so timed that microfilariae escape into the blood stream only at night time with daily death of the cyclic brood. Such an explanation was early explored by MANSON, but rejected on the grounds that the act of

parturition was a continuous process and the filarial rate in the blood and urine did not correspond. Lane now reproduces an interesting graph based on MANSON's original data and points out that, though direct evidence of cyclic parturition is scanty, certain observations, such as those of MANSON-BAHR on the longevity of embryos *in vitro*, and their sudden disappearance following febrile attacks in certain cases, support his hypothesis. The daily disposal of embryos by solution in the blood-stream and visceral filtration with destruction of embryos in the kidney, spleen and liver is discussed, and biological support is adduced from facts concerning the different insect vectors. An optimum insect host and an unspecialized method of parturition are regarded as the original existing types and such is found in Fiji where non-periodicity exists and *A. pseudoscutellaris* is the vector. *C. fatigans* is known to be many times less susceptible than *A. pseudoscutellaris* and it is pointed out that with the former host if the female worm could juggle with the short lived microfilariae by varying the rate of parturition so as to circulate embryos cyclically in the peripheral blood during the few hours when this mosquito bites, then the worm species would survive despite an inefficient intermediary. The author, however, does not regard periodicity as a mere adaptation to the feeding habits of a particular vector ; rather is it an adjustment producing that mean between failure to infect an insect and liability to kill it by hyperinfection which reasonably subserves the survival of the parasitic species.

Manson-Bahr reviews at considerable length LANE's theory of simultaneous cyclical parturition with daily destruction of microfilariae and concludes that there is nothing unbiological in the idea that female filariae have cyclical and well defined periods for emptying the uterus of imprisoned ova. LANE credits *F. bancrofti* with extensive powers of fecundity. In conjunction with Dr. W. K. BLACKIE, the author estimated the amount of blood contained in the peripheral circulation of an adult man of given height, weight, and surface area to be 263 cc. Previous work in Fiji had shown that over 500 embryos were often present in 20 cc. of blood and on this basis the vessels contained in the skin and subcutaneous tissue to a depth of 2 mm. were calculated to harbour over 6,575,000 microfilariae. Not more than 20 adult filariae are found at autopsy, and, in terms of LANE's hypothesis, this would necessitate the daily production of 328,000 embryos per female. The author doubts if any comparative figures on the reproductive capacity in nematodes are available, and also points out that the pathological lesions which should accompany the daily destruction of such myriads of embryos have not been met with.

The survival time of embryos in man is a crucial point in the hypothesis and the pertinent suggestion is made of injecting a volunteer with adequate quantities of *M. bancrofti* under conditions similar to FÜLLEBORN's experiment with *M. repens* in the dog. The author next seeks to fit in certain known facts with cyclic parturition and the different habitats possessed by the two varieties of *F. bancrofti* are considered. In nocturnal *F. bancrofti* the adult worms have a central location, chyluria from obstruction of a main lymphatic trunk being a common complication. The non-periodic *F. bancrofti* of Oceania shows a peripheral habitat in lymph glands and filarial adenitis and elephantiasis are typical complications. Clinically individuals with the highest microfilarial count show fewest signs of disease, a finding

dependent on the uninterrupted passage of embryos into the blood-stream. Microfilariae in the lymph glands must pursue a more complicated route and would reach the blood-stream by a constant trickle both day and night—hence non-periodicity!

Problems connected with *F. loa*, and inversion of periodicity by alteration in sleeping habits, are also dealt with and the author urges a reconsideration of the whole mechanism of periodicity on a wider scale by carefully weighing all the evidence, not only regarding *F. bancrofti*, but other human species as well.

[Serological evidence of massive daily destruction of microfilariae might also be sought for. Circulatory antibody could hardly be absent in the presence of so intense an antigenic stimulus as the one postulated in LANE's hypothesis.]

N. Hamilton Fairley.

O'CONNOR (F. W.) & BURKE (G. R.). **Lymphangitis and Filariasis in Porto Rico. A Report of 57 Cases.**—*Amer. Jl. Trop. Med.* 1929. May. Vol. 9. No. 3. pp. 143–177. [Presbyterian Hosp., San Juan, Porto Rico.]

The authors give descriptions of 47 women and 10 men of whom 17 suffered from lymphangitis without elephantiasis while in the rest the latter was present. Blood examinations (20 cmm. drawn at night) were made in 31, only two containing microfilariae. For lymphangitis an exciting cause was mostly absent, pain was commonly the first symptom, and the condition most commonly showed itself as a small red erythematous patch from which red lines spread up and down the limb. Many experienced recurrences monthly, while in three persons who temporarily left Porto Rico, these occurrences ceased during absence from the endemic area, but re-appeared on return to it. On two, the Kondoleon operation had been performed; one had no benefit, and in the other benefit was temporary.

C. L.

WATANABE (K.). [Klinische und histopathologische Beobachtungen über die Filarialymphangitis des Samenstrangs und über das Lymphom der Inguinofemoraldrüse; insbesondere über die Entstehung der lymphatischen Reaktion und des Sekundärknötchens bei Filariasis.] [**Filarial Lymphangitis of the Spermatic Cord.**]—*Japan Jl. Dermat. & Urol.* 1929. Jan. Vol. 29. No. 1. pp. 1–30. With 15 text figs. [133 refs.] [In Japanese. German summary pp. 1–7.] [Dermat.-Urolog. Clinic, Univ., Nagasaki.]

Seven cases are described in which spontaneously or after exercise there appeared, nearly always without fever, hydrocele and a swelling of the cord unattached to the vas deferens, or of the head of the epididymis and incorporated with it. In six cases the nodule and epididymis were excised. The essential features were dilatation of lymph vessels which contained filarial and amorphous detritus with fragmented nuclei, eosinophils and endothelial cells. The vessel walls were thickened and surrounded by the same cells, mast and plasma cells and a few fibroblasts. There was held to be a new

formation of lymph gland tissue. In another case two cysts were removed from the groin containing filariae and with secondary lymph glands in their wall. It is concluded that lymphatic reaction and secondary lymph gland formation are essential to immunity against filaria. In all cases but one microfilariae were present in the blood.

C. L.

CHOPRA (R. N.) & RAO (S. Sundar). **Studies in the Treatment of Filariasis.**—*Indian Med. Gaz.* 1929. Mar. Vol. 64. No. 3. pp. 130–139. With 2 graphs in text. [6 refs.]

The authors emphasize the finding that the microfilarial content of the blood varies very greatly apart from treatment and give the numbers found in 6 cases, presumably in a fixed quantity of blood. Allowing for this variation no parasiticidal effect could be detected from the use of bisnene, antimosan, stibosan, neo-stibosan, stiburea, novostiburea, antimony sulphur compound, mercurochrome, plasmochin, emetine, or tryparsamide ; though the last produced disappearance of chyluria and decreased the frequency of lymphangitic attacks. This sentence occurs : " In chronic filarial lymphangitis the abdominal glands are usually affected ; the microfilariae do not get into the blood-stream and remain confined to the lymphatics. Many of the parasites subsequently introduced by the mosquito do not mature ; those that mature lay embryos which work back, cause slight lymphatic obstruction and become secondarily infected."

C. L.

CHRISTOPHERSON (J. B.). **The Radioscopical Diagnosis of Filariasis.**—*Brit. Med. Jl.* 1929. May 4. p. 808.

A missionary of 62, who had in his early years of work spent 14 years in which he had frequently been bitten by bugs and mosquitoes without ever suffering from malaria, and who had recently been looking after a leper, began to feel the right leg heavy to move as if it did not belong to him. It was 1½ inches larger than the left in the calf and 2 in. in the thigh. There was eosinophilia of 5 to 9 per cent. Blood and faeces gave no direct evidence of worm infection. X-rays showed in the right calf and thigh, and nowhere else, two or three small oblong cyst-like objects tapering to one end, the shadow ½ in. long, the thick end being lighter.

C. L.

DELBANCO (E.), MÜHLENS (P.) & VOGEL (H.) Bancrofti Invasion mit Chylozele und Periorchitis. [**Case of Filariasis with Chylocele and Periorchitis.**]—*Arch. f. Schiffs- u. Trop.-Hyg.* 1929. Apr. Vol. 33. No. 4. pp. 193–197. With 7 text figs. [Inst. for Ship & Trop. Diseases, Hamburg.]

A negro from Liberia presented an elastic, non-fluctuating, translucent, painless swelling of a testis. Puncture showed a milky fluid with numerous active microfilariae, lymphocytes, red corpuscles and very fine fat particles. The testis, including epididymis, tunica and a length of cord was excised. The last contained a yellowish lymph varix, the hydrocele 30 cc. of chylous albuminous fluid but no adults ; nor did the varix do so, though two pairs of *F. bancrofti* were found in a small lymphatic vessel in the tunica ; this in places was 3 to 6 mm. thick and contained vessels with thickened walls, round cell infiltration and growth of endothelial cells which took on a giant character. There was no local eosinophilia.

C. L.

VINCENZO (Jura). La filariosi in Italia. Un caso di chiluria da Filaria Bancrofti. [**Case of** *F. bancrofti* **Chyluria in Italy.**]—*Policlinico.* Sez. Prat. 1929. June 10. Vol. 36. No. 23. pp. 806–808. With 1 text fig.

No microfilariae were detected in the urine of a man with haemochyluria. Numerous examinations of the circulating blood during sleep displayed on an unstated number of occasions *Mf. bancrofti* which remained mobile for 10 to 15 seconds after being drawn, were as many as 10 to 15 in a single microscopic field, of the diameter of a red corpuscle, 25 to 40 times as long as broad, and the rounded head possessed a rostrum. Vital staining showed a clear external zone and within numerous granules, interspersed with 5 clear or blurred spots. When the urine was filtered immediately after being passed similar bodies were found [presumably in the filtrate]. A photomicrograph is not convincing.

C. L.

NEUBER (Eduard). *Filaria sanguinis hominis* (*Filaria bancrofti*). Vorläufige Mitteilung. [**Case of** *Filaria bancrofti* **Infection.**]—*Wien. Klin. Woch.* 1929. July 11. Vol. 42. No. 28. pp. 937–938.

A man of 57, inhabitant of north Hungary, had during the war been stationed in Bohemia, and had never been elsewhere. He developed fever with rigors and ended up with an elephantoid scrotum. Larvae of *Filaria bancrofti* were found in the blood, sheathed, 300 $\mu$ long, with sinuous movements and having a central core of nuclei.

C. L.

CORT (W. W.), OTTO (G. F.) & SPINDLER (L. A.). **Studies on Ascariasis in Virginia. A Preliminary Report.**—*Southern Med. Jl.* 1929. July. Vol. 22. No. 7. pp. 608–613. [5 refs.] [School of Hyg. & Public Health, Johns Hopkins Univ., Baltimore.]

Distribution and intensity of infection were measured by Stoll's egg counting method, the degree of visible soil pollution by inspection, and the amount of ascaris infection of the soil by indirect centrifugal floatation in a solution with a specific gravity of 1350 (obtained by bichromate of soda solution), the surface being then examined as in the Kofoid and Barber technique. There was no racial difference between whites and negroes in the distribution of infection, which was definitely familial. The writers definitely speak of ascaris families, and from the soil about their houses, or more frequently under them when they were raised at one end from the sloping hillside, ascaris eggs, often embryonated, were commonly found. Under these houses the younger children commonly played, it was the younger children under school age who were commonly or even solely infected, and it was they who did not use the latrines either because of the difficulty of climbing on to the seat or of the fear of falling through the hole when they did. Indeed this seems a very justifiable fear in view of FLANNAGAN'S statement in discussion that children, he was told, had been drowned in "septic privies." At least in present local conditions these children are the main source of infection. Recommendations include a "children's hole" and school education which may in the next generation be as effective in securing its object as it has been in the past in obtaining prohibition in America.

C. L.

BORCHARDT (W.). Zur mutmasslichen Genese der Eosinophilie bei Ascarisinfektion und evtl. auch bei anderen Wurmkrankheiten. I. Mitteilung. Die Wirkung von aliphatischen Aldehyden. [**The Probable Origin of Eosinophilia in Ascaris Infection.**]—*Klin. Woch.* 1929. Mar. 26. Vol. 8. No. 13. pp. 591–594. [5 refs.] [Inst. for Ship & Trop. Diseases, Hamburg.]

FLURY [*Arch. f. exper. Path.*, 1912, Vol. 67, p. 275] stated that aldehydes of the fatty acids were found in ascaris. Borchardt injected these substances into cats and found that in small doses they produced eosinophilia (except acetaldehyde), and in larger doses an increase in polymorphs. The other compounds used were prophylaldehyde, crotonaldehyde, formaldehyde, isobutyraldehyde and isovalerylaldehyde.

C. L.

SCHWEIZERISCHE MEDIZINISCHE WOCHENSCHRIFT. 1929. May 11. No. 19. pp. 493–494. Enquête ueber Pathogenität und Verbreitung der Askariden in der Schweiz. Die Bedeutung der Spulwürmer in der menschlichen Pathologie [ASKANAZY]. Technik der Gewinnung und mikroskopischen Untersuchung auf Helminthen-Eier und Larven [GALLI-VALERIO]. [**Ascaris Infestation in Switzerland. Examination for Ova.**]

Askanazy describes various ill-effects which ascaris has produced either as adult or larva. Galli-Valerio describes microscopic diagnosis either by 5 covered preparations of a faecal suspension in water or in 1 to 2 per cent. formalin solution, or by floatation in 37·5 per cent. salt solution in an Erlenmeyer flask for 5 to 45 minutes and examination of the surface layer. Lettuce leaves are cut up small, soaked in water, then sedimented by gravity for 15 to 20 minutes and the sediment examined. It is necessary to examine 40 or 50 preparations before a negative conclusion can be accepted. [In the abstracter's counts floatation in an Erlenmeyer flask has delivered on the average 2 per cent. of hookworm ova placed within it. The present statements are uncontrolled.]

C. L.

BOETTIGER (Carl) & WERNE (Jacob). *Ascaris lumbricoides* **found in the Cavity of the Human Heart.**—*Jl. Amer. Med. Assoc.* 1929. July 6. Vol. 93. No. 1. p. 32. [St. John's Long Island City Hosp., Long Island City.]

A woman with ulcerated elephantiasis, and an acute illness of a few hours' duration, showed post-mortem pulmonary thrombosis and chronic interstitial pneumonia. "The interesting feature of this case was the finding of two yellowish white worms with annular markings, measuring each about 10 cm. by 5 mm., in the cavity of the right ventricle. . . . No adult forms of the parasite were found in the intestine. . . . The worms were identified as forms of *Ascaris lumbricoides*." [An expert's identification of these annulated bodies will be awaited with interest.]

C. L.

DIRCKS-DILLY (J.). Deux cas de néphrite par ascaris dont un suivi de mort. [**Two Cases of Ascaris Nephritis, One Fatal.**]—*Gaz. hebd. Sci. Méd. de Bordeaux.* 1929. Mar. 24. Vol. 50. No. 12. pp. 186–187.

The fatal case, a recruit, weighed only 47·5 kilos, had erythema, fever, vomiting, right hypochondriac pain suggesting appendicitis and more vomiting with passage of four ascarids. Thymol administered had apparently no vermifugal effect, melaena and arthritis appeared, and in the third week albuminuria and later, casts. Ascaris and trichuris eggs persisted and he died with evidence of intense toxaemia. No autopsy is mentioned. The second, also a soldier, had oedema and urine with albumin and a specific gravity of 1011. Ascaris and trichuris eggs were present, but were no longer formed after chenopodium. The nephritis was not, however, cured. The writer answers in the positive the question he puts: "Is there ascaris nephritis?"

C. L.

DSCHAPARIDSE (P.). [Ein Fall der starken Askarideninvasion mit schweren klinischen Erscheinungen.] [**Case of Ascariasis with Severe Clinical Symptoms.**]—*Nachrichten d. Tropischen Medizin.* Tiflis. 1928. Dec. Vol. 1. No. 2. [In Georgian script. German summary p. 225.]

A girl of 16 had ailed for three years with weakness, pain in the head and abdomen, loss of memory and sleeplessness. Latterly she had been bed-ridden with weakness. Anaemia was marked and unrelieved by quinine. Eggs of ascaris, oxyuris and trichuris were present. Santonin caused a week-long exodus of ascaris by mouth and anus, she left her bed in a fortnight, and was fully recovered in three months.

C. L.

DIXEY (M. B. Duncan). **A Case of Sudden Death due to the Occlusion of the Larynx by Two** *Ascaris lumbricoides.*—*Edinburgh Med. Jl.* 1929. Feb. Vol. 36. No. 2. p. 111.

A negro girl of 8½ years died within four or five minutes of leaving her mother well. Two large ascarids completely occluded the larynx, one being partly curled up in it. Another ascaris lay in the stomach and two more in the small intestine. There were the usual bodily signs of asphyxia. It is suggested that increased respiration (she was carrying water back from a well) caused inspiration of worms which had reached the pharynx.

C. L.

STOLL (Henry F.). **Trichinosis. Report of Two Cases presenting Diplopia and One, Polyserositis.**—*Jl. Amer. Med. Assoc.* 1929. Mar. 9. Vol. 92. No. 10. pp. 791–793. With 2 text figs. [8 refs.] [Hartford Hosp., Hartford, Conn.]

In the first two cases ocular symptoms (extreme conjunctival oedema, diplopia) accompanied a marked eosinophilia. Excised portions of deltoid and trapezius showed trichinella larvae of which photographs are shown. The third patient developed pleurisy and pericarditis, and showed eosinophilia of 21 to 31 per cent. and a precipitin reaction with 1 in 4,000 serum dilution, but no trichinae were found in excised trapezius. It is considered possible that infection had occurred on an occasion 18 years earlier, when he had had pain in the legs and thighs so severe that he took ¾ of an hour to get to the telephone and back down two

flights of stairs, to summon a Christian Science healer. The pain however, lasted several weeks under his treatment and, most inopportunely, recurred several months later when the patient was acting as usher at a Christian Science meeting, and was so severe that in the middle of the lecture he had to lie down on the floor.

C. L.

DE LANGEN (C. D.). **Anguillulosis and the Syndrome of the "Idiopathic Hypereosinophilia."**—*Meded. Dienst d. Volksgezondheid in Nederl.Indië.* 1928. Vol. 17. Pt. 4. pp. 515–529. [Dept. of Internal Diseases, Med. Univ., Batavia.]

In 1923 de Langen published 3 cases of "idiopathic hypereosinophilia." These are briefly redescribed [see this *Bulletin*, Vol. 21, p. 499.] and sixteen others are added. These cases gave a history of 4 months to ten years illness, and showed a leucocytosis with eosinophils as high as 85 per cent., and a syndrome of slight anaemia, epigastric pain and heaviness, intermittent diarrhoea, slight rises in temperature, and pulmonary symptoms—cough, mucopurulent expectoration, an asthmatic condition and broncho-pneumonic foci shown scattered through the lungs under X-rays. No tubercle bacilli were found in spite of daily examination for weeks. Strongyloides infection was considered as a cause, the sputum examined, and "finally we succeeded in finding therein a few larvae." Accordingly regular examinations were made of the faeces of an unstated number of persons and Strongyloides larvae were found in 26 by smear, or by culture for 24 hours with subsequent extraction by Baermann's method. In this group of twenty six were found 12 of the 16 hypereosinophilias already mentioned; the other persons were in hospital for other causes and had eosinophilia varying from 3 to 29. Treatment was undertaken by tartar emetic intravenously which cleared up the general condition but left a fair intestinal infection. Accordingly gentian violet by mouth 0·1 to 0·3 gm. three to five times daily was added. Although an occasional larva could sometimes still be found, the morbid symptoms and eosinophilia disappeared in a most striking way. [The X-ray condition of the lungs after clinical cure seems nowhere stated. The findings suggest a pulmonary habitat for a proportion of the parthenogenetic forms.]

C. L.

ST. JOHN (Joe H.), SIMMONS (James S.) & GARDNER (Leon L.). **Infestation of the Lung by a Nematode of the Genus Cyathostoma.**—*Jl. Amer. Med. Assoc.* 1929. June 1. Vol. 92. No. 22. pp. 1816–1818. With 3 text figs. [2 refs.] [U.S. Army Med. Dept. Research Board, Bureau of Science, Manila, P.I.]

Three days after returning to Manila from a climbing expedition, a man developed acute bronchitis, passing into a chronic illness with paroxymal cough, asthma, mucopurulent sputum often streaked with bright blood, pain and tenderness on palpation and percussion in the first left intercostal space 1 in. from the sternum. The sputum showed colourless thin-shelled ova 102$\mu$ by 60$\mu$, oval and generally flattened on one side and containing 8 cells and therefore fertile. An eosinophilia

varied from 7 to 28 per cent. Treatment was unavailing, but one day a moderate attack of coughing expelled a blood-red female nematode, 16·6 mm. long. The oral cavity " contained at its base, seven teeth, and a fleshy tooth-like projection which probably carried a glandular duct." The vulva lay 3·52 mm. from the head. [The description of the internal organs does not seem to show specialist knowledge. *Cyathostoma* E. Blanchard, 1849 is a synonym of *Syngamus* Siebold, 1836.]

C. L.

HEYDON (G. M.). **Creeping Eruption or Larva Migrans in North Queensland and a Note on the Worm** *Gnathostoma spinigerum* **(Owen).**—*Med. Jl. Australia.* 1929. May 4. 16th Year. Vol. 1. No 18. pp. 583–591. [19 refs.] [Australian Inst. of Trop. Med., Townsville.]

Oestrid larvae, immature gnathostomes and as CILENTO has shown for Rabaul [this *Bulletin*, Vol. 25, p. 290], the mite Rhizoglyphus cause creeping eruption, as well as nematode larvae. The last cause is not uncommon about Townsville in the wet hot summer in those who go barefoot either to the sea, or to the shower bath beneath the pile-raised houses where the ground is slightly moist and a resort of dogs and cats. These animals are nearly always infected with *Ancylostoma caninum* and *A. braziliense*, as many as 552 hookworms having been found in one cat. Experimental infections in man produced in some cases creeping eruption and that with larvae both of *A. caninum* and *A. braziliense*, but in no case did D.C.F. show subsequent intestinal infection. Larvae of *Trichostrongylus colubriformis* produced no local effect on the skin and no infection of the bowel. The itchiness accompanying a creeping eruption is often [as Looss pointed out in his own person] localized to a point a few millimetres beyond the advancing end of a track and gives the impression of something moving ; but Heydon adds the observation that " close inspection of such a spot will sometimes reveal a tiny elevation and that the track is really continuous right up to it, though for the last few millimetres it is only a very inconspicuous mark in the epidermis."

In view of FERGUSON's statement [*l.c.* Vol. 25, p. 700 : G. R. HAMILTON] that he knew of no record of gnathostomes in Australia, Heydon reports the not infrequent finding of *G. hispidum* in Rabaul in the stomachs of pigs largely of the kanaka breed with some admixture of imported stock. He also found *G. spinigerum* in a cyst in the stomach wall of 1 of about 40 cats examined. It contained 19 males and 30 females. The lumen of cyst, stomach and rectum contained eggs in numbers more or less equal to those derived from 227 hookworms also present. These eggs were nearly all one-celled, some two-celled. It thus appears that the intra-uterine embryonated eggs reported by Clayton LANE from the Calcutta cat " possibly arose from the examination of worms preserved in a manner which allowed the eggs to continue their development " [the interval between the host's death and the worm's preservation was, however, unknown.] Development occurred well at summer room temperature, but that of 37° C. delayed or prevented development, or killed eggs. Spontaneous hatching is very rare. Newly hatched larvae 0·25 mm. long lie in a voluminous, unstriated, delicate sheath.

C. L.

CAUCHEMEZ (L.). Un cas remarquable d'appendicite à oxyures. [**Remarkable Case of Appendicitis with Oxyuris.**]—*Ann. Parasit. Humaine et Comparée.* 1929. July 1. Vol. 7. No. 4. pp. 280–281. With 4 figs. on 2 plates. [Lab. of Parasit., Faculty of Med., Paris.]

An inflamed adherent appendix removed from a child of 8, showed on section numerous portions of enterobius lying within the mucosa and in the lumen. There had before operation been nothing to suggest that the child was parasitized with threadworm.

C. L.

SCHULZ (Richard Ed.). [*Trichostrongylus orientalis* (Jimbo, 1914) als Parasit des Stachelschweins.] [*Trichostrongylus orientalis* **as Parasite of Porcupine.**]—*Rev. Microbiol., Epidémiol. et Parasit.* 1929. Vol. 8. No. 1. pp. 45–48. [11 refs.] [In Russian. German summary pp. 115–116.]

The porcupine is implicated as a "reservoir host" of *Trichostrongylus orientalis.*

C. L.

BOLETIN DEL INSTITUTO DE CLÍNICA QUIRÚRGICA. Buenos Aires. 1927. Vol. 3. Nos. 21–25. pp. 147–191.—II. Uncinariosis. [**Uncinariasis.**] [Also issued as *3a Reunión, Soc. Argentina Patol. Regional del Norte, Tucumán, Julio 7, 8 y 10, 1927.* pp. 39–83.]

In Section II of the report of the Third Session of the *Sociedad de Patologia Regional del Norte* are contained eight articles treating of helminthic problems. Two, of a more strictly scientific character from the helminthological aspect, are by Drs. SOPER and VOGELSANG; the first comparing *A. duodenale* and *N. americanus* from the point of view of oviposition, the second comparing and differentiating *A. brasiliense* and *A. caninum.* By calculations based, unfortunately, on a very small number of cases Dr. SOPER found that the daily output of *A. duodenale* was from two to two and a half that of Necator, and the range of variation practically the same. He concluded that the variation was not the result of treatment, but was probably a specific peculiarity or due to cyclical production of ova [see this *Bulletin,* Vol. 24, p. 1006].

The differences relied upon by Dr. VOGELSANG for distinguishing *A. brasiliense* from *A. caninum* were a well-marked triangular tooth on each side of the buccal capsule and a pair of accessory teeth at the anterior superior angle in the latter. In a dog dying in Tabacal (Salta) nine specimens of *A. caninum* were found and three of *A. brasiliense,* while in another at San Pedro (Jujuy) only the former were present.

The succeeding four papers are of an epidemiological character. The first is a study of the infection in the Departments of San Pedro and Ledesma. Of 233 school-children examined 22·31 per cent. were found infected [no details of technique]. The figures obtained at the six different schools are not of much value because the numbers are so small; at none were more than 45 examined and at one only twenty. Further investigations are recorded in another article dealing with San Pedro where infection is both severe and widespread. In some parts, Corrientes, for example, the percentage was 60 and even higher. There the people were living under very primitive and unhealthy

conditions in adobe huts with an earth floor, no latrines, and the dwellings occupied also by several animals. Most of the inhabitants showed obvious signs of infection—pallor, oedema, wasting of muscles, palpitation, languor and general debility. In many the erythrocyte count was down to a million and the haemoglobin to 25 per cent. or less. Mass treatment is being undertaken.

The western part of the city of Tucumán was also found to be an endemic focus; the general appearance of many individuals was suggestive of the disease and of 70 whose faeces were examined 42 proved positive. In the Northern Argentine, again, infection is heavy; thus at Chaco, 30 leagues west of Concepción, every one examined [the number is not stated] was found to be harbouring *A. duodenale* and 50 per cent. of them had *N. americanus*. The relative proportion of the worms was 13 to 1.

The last two papers are concerned with anthelmintic plants. Several of these, or at least plants having such reputation, are indigenous to the country; the following are the chief: *Aspidium capense*, having an action similar to that of Filix mas; *Ficus anthelmintica*, *F. cestrifolia*, *Carica papaya*, *Jaracatia dodecaphylla*, and, finally, two varieties of Chenopodium, *C. ambrosioides* and *C. anthelminticum*.

H. Harold Scott.

Garin (Ch.), Doubrow & Mounier. Sur la possibilité de developpement des larves dans l'utérus de la femelle morte d'ankylostome. Conséquences thérapeutiques et prophylactiques. [**Development of Larvae in Uterus of Dead Female Ankylostome.**]—*Lyon. Méd.* 1928. June 3. Vol. 141. No. 23. pp. 634–635. With 4 figs. on 1 plate.

When female hookworms are washed in saline and kept at a warm temperature for 24 hours numerous larvae develop and feed upon the internal organs. The authors think that a similar development may be probable in the human body and deduce therefrom the possible invasion of the intestinal mucosa by the infective larvae. This would afford an explanation of the difficulties which are sometimes encountered in the eradication of hookworm infestations. [It is stated that this phenomenon has only been noticed previously by Alessandrini but it is a common technique for the cultivation of pure strains of larvae of bursate nematodes in agricultural parasitology.]

R. T. L.

Chandler (Asa C.). **The Rates of Loss and Acquisition of Hookworms.** —*Jl. Amer. Med. Assoc.* 1929. Apr. 20. Vol. 92. No. 16. pp. 1337–1341. With 2 charts in text. [13 refs.]

Chandler considers the evidence which has been offered regarding the rate at which hookworm infection is got and lost particularly with reference to Indian work, that of Mhaskar (*Indian Jl. Med. Res.*, Vol. 8, p. 398) and himself (this *Bulletin*, Vol. 23, p. 773). While the degree of infection in an Indian jail had lessened by 92 per cent. in 5 years, the number of infected persons had not. Diagnosis was in this case by the Kofoid and Barber technique and the counts of eggs by Chandler's modification of Stoll's technique. Further detailed studies are urged.

C. L.

VILLELA (Gilberto G.) & TEIXEIRA (J. Castro). [In Portuguese & English.] Exame do sangue na anemia helminthica. **Blood Study in Hookworm Anemia.**—*Inst. Oswaldo Cruz. Supplemento das Memorias.* 1929. Mar. No. 6. In Portuguese pp. 55–61. In English pp. 62–68. [11 refs.]

Very few biochemical observations have been made on the blood serum in hookworm disease. In patients with ankylostomiasis studied in Rio de Janeiro it was found that the blood serum calcium was normal or reduced, while the potassium content was generally low (16·5 to 22 mgm. per 100 cc. of serum), possibly as a result of the fall in the number of red cells. The chlorides were increased, the total protein content reduced and the cholesterol normal in amount. Anthelmintic treatment was followed by a return to the normal in all the blood constituents studied.

G. M. Findlay.

SCOTT (J. Allen). **Experimental Demonstration of a Strain of the Dog Hookworm,** *Ancylostoma caninum,* **especially adapted to the Cat.**—*Jl. Parasit.* 1929. Mar. Vol. 15. No. 3. pp. 209–215. [19 refs.] [School of Hyg. & Public Health, Johns Hopkins Univ., Baltimore, Md.]

The author reviews the varying relative parasitization of dogs and cats with *Ancylostoma caninum* in various parts of the world, and notes that in certain places it is absent from cats though common in dogs. Accordingly he has undertaken experiments with two strains, one from dog and one from cat. All host animals were kept long enough to eliminate previous immature infections and all were found negative by D.C.F. " This method will indicate the presence of a single mature [fertilized] female." Larvae from charcoal cultures were given orally in gelatin capsules, which practically eliminated any tendency to vomit, to pups and kittens kept in individual cages, controls kept in identical conditions remaining uniformly uninfected. When cat strain was fed to kittens 45 per cent. of larvae matured, when to pups 0·4 per cent. Conversely when dog strain was fed to pups 50 per cent. matured, when to kittens 4·6 per cent. There is doubt regarding the last number since this kitten had to be returned to a single cage for two days and the control, while showing no adult hookworms, showed 26 undeveloped larvae to Baermann's larval extraction apparatus. It is concluded that the experiments demonstrate two strains of a parasite morphologically identical but physiologically different in their adaptation to different hosts.

[It would be valuable to obtain information as to any effect on dermal infection of such host influence.]

C. L.

YOKOGAWA (S.). [**Occurrence of** *Ancylostoma braziliense* **Gomez de Faria in the Aborigines of Formosa and in their Cat.**]—*Taiwan Igakkai Zasshi* (*Jl. Med. Assoc. Formosa*). 1929. May. No. 290. [In Japanese. English summary p. 23.]

*Ancylostoma braziliense* was found in a cat caught in a mountain district of N. Formosa. The aborigines could not be induced to undergo anthelmintic treatment. Faeces from them were cultured and fed to a cat in

whom later an adult female of this species was found. No mention is made of efforts to prove that the cat was originally free of infection or did not become naturally infected later.

C. L.

DSCHAPARIDSE (P.). [Hakenwurmkrankheit in Abchasien und ihre Bekämpfung.] [**Hookworm Disease in Abkhasia and its Prevention.**]—*Nachrichten d. Tropischen Medizin.* Tiflis. 1928. Vol. 1. No. 1. [In Georgian script. German summary p. 112.]

By diagnostic methods unstated in the German abstract it is concluded that 75 per cent. of children suffer from ankylostomiasis, *Necator americanus* being the predominant hookworm. Mass treatment with thymol was carried through.

C. L.

FRANCHINI (G.) & BACCHELLI (G.). Su di un focolaio di Anchilostomo-anemia in Provincia di Modena. [**Ankylostomiasis in Modena.**]—*Arch. Ital. Sci. Med. Colon.* 1929. Feb. 1. Vol. 10. No. 2. pp. 53–58. With 2 text figs. English summary p. 58. [Inst. of Trop. Path., Univ., Bologna.]

A young man of 17 years came to the medical clinic of Modena. He was anaemic and his blood contained 13 per cent. eosinophiles. Faecal examination revealed ankylostome ova. Further examination of his parents, two brothers, and two sisters proved that the whole family was infected.

H. Harold Scott.

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KARIBOFF (N.). [Données préliminaires sur l'invasion vermineuse dans quelques groupes de la population de Tiflis.]—*Russian Jl. Trop. Med.* 1929. Vol. 7. No. 1. pp. 45–46. [In Russian.]

KIRBY-SMITH (J. L.), DOVE (W. E.) & WHITE (G. F.). Some Observations on Creeping Eruption.—*Amer. Jl. Trop. Med.* 1929. May. Vol. 9. No. 3. pp. 179–192. With 1 plate. [11 refs.]

MARIA MACERA (Jose) & CARRENO (Carlos). La entozoosis en clinica pediatrica.—*Cronica Med.-Quirurg. de la Habana.* 1929. Feb. Vol. 55. No. 2. pp. 81–88.

NICHOLSON (Daniel). Variations in the Fish Larvae of *Diphyllobothrium latum.*—*Canadian Public Health Jl.* 1929. Apr. Vol. 20. No. 4. pp. 193–195. With 1 text fig. [1 ref.]

SCRIABINE (K. I.), PODYAPOLSKAYA (V. P.) & SCHOULTZ (R. S.). [L'oeuvre de la 60-me expédition helminthologique en l'Extrême Orient Sibérien.]—*Russian Jl. Trop. Med.* 1929. Vol. 7. No. 1. pp. 36–44. [In Russian.]

STUNKARD (H. W.). Observations nouvelles sur les trématodes sanguicoles du genre *Vasotrema* (*Spirorchidae*) avec description des deux espèces nouvelles.—*Ann. Parasit. Humaine et Comparée.* 1928. July. Vol. 6. No. 3. pp. 303–320. With 9 text figs. [19 refs.]

STUNKARD (Horace W.). The Parasitic Worms collected by the American Museum of Natural History Expedition to the Belgian Congo, 1909–1914. Part I.—Trematoda.—Reprinted from *Bull. American Museum Natural History.* 1929. Apr. 15. Vol. 58. Art. 6. pp. 233–289. With 37 figs.

WARNOWSKY (I.). Ueber endemische Infektion durch Trichocephalus dispar im Memelgebiet und Gross-Litauen.—*Muench. Med. Woch.* 1929. July 12. Vol. 76. No. 28. p. 1171.

ZDRODOWSKI (P.). Ankylostomose.—Institut de Microbiologie et d'Hygiène d'Azerbaidjan. Bakou. 1929. 60 pp. [In Russian.] With 1 map & 3 plates.

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## YELLOW FEVER.

PUBLIC HEALTH REPORTS. 1929. July 12. Vol. 44. No. 28. pp. 1657–1659. [2 refs.]—**Yellow Fever at Rio de Janeiro.**

During 1928 yellow fever reappeared at Rio de Janeiro [*ante*, p. 294] and up to October 6th, 119 cases were reported with 66 deaths. From that date until January 12th, 1929, there were only 7 cases, with 4 deaths; but beginning in January the prevalence increased and up to June 1st, 1929, there have been reported 615 cases, with 351 deaths. The results indicate that the yellow fever curve rises during the summer months when conditions are most favourable for the life and multiplication of the mosquitoes. The present outbreak extended to all parts of the city, from the dense rural zones out to remote suburbs; consequently the difficulty of anti-mosquito measures was much increased. About 6,000 men a day are being employed in the campaign against mosquitoes. In addition to the destruction of larvae, more than 400 houses a day are being disinfected by sulphur or insecticides. The most commonly used insecticide is a mixture of petroleum, carbon tetrachloride and pyrethrum. In addition, all cases are isolated and a careful sanitary supervision is made of the sections where any have occurred, following the radius of action of the mosquito carrier.

E. Hindle.

HANSON (Henry). **Observations on the Age and Sex Incidence of Deaths and Recoveries in the Yellow Fever Epidemic in the Department of Lambayeque, Peru, in 1921.**—*Amer. Jl. Trop. Med.* 1929. July. Vol. 9. No. 4. pp. 233–239.

An interesting account of the last yellow fever epidemic in Peru in 1921. A list is given of the age and sex of 478 cases of the disease, which clearly shows that infants of 1 and 2 years old are particularly susceptible, and that the great majority of the cases occurred in people under 30 years of age. The explanation given is the fact that the last epidemic in Peru was 25 years previously, and consequently the majority of the population above this age is immune. The records show that practically every patient more than 25 years old entered Peru after the previous epidemic.

The highest death rates occur among infants especially those between 1 and 3 years of age, and in persons of 5 years or older the lowest death rate is between 11 and 15 years. The death rate in males is 2·5 per cent. higher than in females [but the figures are not large enough to be of significance]. Deaths are recorded on the 2nd to the 24th days of illness, but the majority occurred on the 6th day, which seems to be the day of crisis.

[The author's observations as to the susceptibility of infants are opposed to the view, sometimes advanced, that the disease is maintained in endemic centres owing to infants becoming infected without showing any clinical signs of yellow fever.]

E. H.

DE MELLO (E. Jansen). Indices de mosquitos e outros indices em campanhas anti-amarillicas. [**Indices of Importance in Yellow Fever Campaigns.**]—*Folha Med.* 1929. June 15. Vol. 10. No. 17. pp. 197–203.

This is a valuable article but one which unfortunately cannot be abstracted and must be consulted in the original. It deals with statistical methods in general, and proceeds to demonstrate these methods by special application to mosquito prevalence and breeding sites and other matters of importance in yellow fever and the various factors which influence them. As a running commentary it points out also the dangers of compilation of statistics by persons who are not fully alive to the pitfalls which beset their path.

H. Harold Scott.

CAZANOVE (F.). Le diagnostic prophylactique et les symptômes de début de la fièvre jaune. [**Prophylactic Diagnosis and Symptoms in the First Stages of Yellow Fever.**]—*Bull. Soc. Path. Exot.* 1929. June 12. Vol. 22. No. 6. pp. 447–473.

The author in this long and detailed account insists on the importance of an early diagnosis of yellow fever and discusses the best methods of recognizing the disease. A negative examination for blood protozoa (malaria, etc.) combined with the presence of albumin in the urine is considered a sufficient indication of the disease, especial weight being given to the latter symptom. The paper is accompanied by details of numerous cases illustrating the points considered by the author.

In the discussion Prof. MARCHOUX gave his opinion that yellow fever epidemics often start with very mild cases which are not recognized until after mosquitoes have had the opportunity of becoming infected. Continuous anti-mosquito measures seem to be the only certain method of preventing the return of epidemics.

E. H.

LINS (Sinval A.). Contribuição ao estudo clinico da febre amarella. [**Contribution to the Clinical Study of Yellow Fever.**]—*Archivos de Hyg.* Rio de Janeiro. 1929. Vol. 3. No. 1. pp. 195–404. With 33 charts. [Refs. in footnotes.]

Long though this article is, it is not lengthy, it is worth reading more than once, and should be reproduced in English so as to reach a wider circle. Here it is only possible to intimate the outlines of the article. The author divides cases into three main groups: abortive, mild renal, and hepato-renal with albuminuria, haemorrhages and jaundice. This last group he again divides into three grades, benign, severe, and hypertoxic, and seven further subdivisions according to the predominance of certain special symptoms. [From the point of view of a clinical *study* this is valuable, but in practice it would hardly be possible to relegate many cases under one or other head.] Cases are reported in detail to illustrate the author's points. He next considers special symptoms, such as temperature, pulse, results of blood examination, of the van den Bergh test, and of the urine, and gives his interpretation of the results and of the pathogenesis of the main symptoms. The

blood examinations comprise the morphological changes, the coagulability and the chemical findings, and from these he discusses the metabolic changes in the body. A special section is given to the "pathogenesis of haemorrhages." A few words are added on treatment, though the author aptly sums the matter up thus: "Yellow fever is a disease which either gets well of itself or kills in spite of all treatment." He hopes to deal with this side of the question more fully on a future occasion.

H. Harold Scott.

OTERO (Francisco). Fiebre amarilla. Exposición analitica de los sintomas en relación con el diagnóstico respectivo. [**Yellow Fever. Consideration of the Symptoms as of Diagnostic Import.**]—*Semana Méd.* 1929. June 6. Vol. 36. No. 23 (1847). pp. 1463–1471.

The author discusses the chief symptoms seriatim and shows that none is diagnostic alone, but that the general symptom-complex is fairly stable, the most serviceable being the early and progressive albuminuria. He concludes in the following words: "A patient coming from an infected locality, whether jaundiced or not, whether showing haemorrhages or not, vomiting or not vomiting, but whose condition is that associated with a toxic process of rapid evolution [typhoid state] following a marked general reaction and with much albumin in the urine, should be regarded with grave suspicion."

H. Harold Scott.

CHAGAS (Evandro) & DE FREITAS (Lincoln). [In Portuguese & English.] Electrocardiogramma na febre amarella. **Electrocardiogram in Yellow Fever.**—*Inst. Oswaldo Cruz, Supplemento das Memorias.* 1929. May. No. 7. In Portuguese pp. 72–78. In English pp. 79–85. With 33 figs. on 8 plates.

A study of variations in electrocardiograms of eleven cases of yellow fever, eight of which were fatal. Although the changes are not very characteristic, the cardiac muscle degeneration is registered very accurately, and may give an indication of some value for prognosis.

E. H.

FERRARI (Antonino). A urologia na febre amarella. (Estudos realizados no Hospital São Sebastião). [**Urinalysis in Yellow Fever.**]—*Brasil-Medico.* 1929. May 18. Vol. 43. No. 20. pp. 551–559.

Examination of the urine at different stages of yellow fever has led the author to the following conclusions regarding diagnosis and prognosis:—

1. Albumin in the first three days of fever, with a negative diazo reaction, is diagnostic of the disease. The amount of albumin is of no prognostic significance.
2. The albumin increases in the second stage, but clears up rapidly with convalescence. Chronic nephritis is rare as a sequela.
3. A large quantity of urates in the first or second stage is a favourable indication.

4. Glycosuria is not met with. The degenerative lesions of the liver, pancreas, and kidneys account for the slow elimination of glucose.
5. The increased acidity calls for the use of alkalies, of which sodium bicarbonate is best.
6. Oliguria is constant from the second to the fourth days.

H. Harold Scott.

Le Boucher (H.), Capéran (G.), Laigret (J.) & Tchernenko (A.). Note sur un cas d'ictère à vomissements noirs. [**Note on a Case of Jaundice with Black Vomit.**]—*Bull. Soc. Path. Exot.* 1929. July 10. Vol. 22. No. 7. pp. 612–618. [2 refs.] [Hosp., & Vaccine & Biol. Lab., Bamako.]

The record of an obscure case at Bamako in which the patient, a French soldier, died with symptoms closely resembling yellow fever, including jaundice and black vomit. Examinations for blood protozoa and spirochaetes, and also blood cultures gave negative results, as did inoculations into guineapigs and a Cynocephalus monkey. This was an isolated case, no others being observed amongst the other soldiers or the natives of the vicinity, in spite of the presence of *Aëdes aegypti*. The nature of the case was not decided.

E. H.

Torres (C. Magarinos). Sur les inclusions nucléaires dans la fièvre jaune expérimentale (virus brésilien et africain). [**The Nuclear Inclusions in Experimental Yellow Fever (Brazilian and African Strains).**]—*C.R. Soc. Biol.* 1929. July 17. Vol. 101. No. 24. pp. 951–952. [1 ref.]

Further investigation of these intranuclear bodies [*ante*, p. 648] shows that they are most evident in monkeys inoculated with the Brazilian strain on the 6th to 10th days after inoculation and in those which die earlier the bodies may be absent. After recovery the inclusions gradually disappear. In one case, however, they were found in the liver of a monkey which died 36 days after inoculation. Thirteen monkeys inoculated with the African virus, which all died in 3 to 9 days, all showed the presence of these inclusions, which evidently develop much more rapidly with this strain than with the Brazilian one.

E. H.

Torres (C. Magarinos). Altérations nucléaires des cellules du foie chez les singes inoculés avec les virus brésilien et africain de la fièvre jaune. [**Nuclear Alterations in the Liver Cells of Monkeys inoculated with Brazilian and African Strains of Yellow Fever.**]—*C.R. Soc. Biol.* 1929. July 17. Vol. 101. No. 24. pp. 959–961. With 1 text fig. [2 refs.]

Although these intranuclear bodies were found in five monkeys infected by the inoculation of blood from patients, they were not found in the livers of twelve fatal cases of the disease in human beings. The author considers that the disappearance of the virus from the blood and organs before death is an explanation of the absence of the bodies in these cases.

E. H.

HOFFMANN (W. H.). Einschlüsse in den Leberkernen beim menschlichen Gelbfieber. [**Intranuclear Inclusions in the Liver of Human Cases of Yellow Fever.**]—*Arch. f. Schiffs- u. Trop.-Hyg.* 1929. Aug. Vol. 33. No. 8. pp. 411–413.

The author re-examined a large number of his old sections of human yellow fever livers, and in only one instance were intranuclear bodies found. These were in the liver from a West African case of the disease, which from its general appearance suggested a very acute attack. The author is of the opinion that the intranuclear bodies are the result of nuclear degeneration, but it remains to be discovered whether the yellow fever virus is responsible for their appearance, or is possibly concealed by them.

[It is of interest that TORRES [see above] failed to find these bodies in human cases of the disease, and HOFFMANN's results agree with the view that they are very rarely present at death, by which time the virus has usually disappeared from the body.]

E. H.

FIALHO (Amadeu). Contribuição ao estudo da anatomia pathologica da febre amarella. Primeira memoria. Diagnostico anatomico. [**A Contribution to the Pathological Anatomy of Yellow Fever.**]—*Archivos de Hyg.* Rio de Janeiro. 1929. Vol. 3. No. 1. pp. 37–91. With 5 coloured plates. English summary facing p. 90.

The author performed some scores of autopsies on yellow fever cases in Rio de Janeiro, and gives a summary of the main features in their gross pathology, which agree with those that have been described in other epidemics. The paper contains five good coloured plates illustrating respectively the surface of the heart, the lining of the stomach, the tongue and posterior buccal cavity, sections of the kidney and liver, and finally a section of another kidney and the surface of a lobe of the liver.

E. H.

MONTEIRO (J. Lemos). Estudos sobre a febre amarella. Sobre a possibilidade de um diagnostico bacteriologico da febre amarella. Nota prévia. [**Bacteriological Diagnosis of Yellow Fever.**]—*Brasil-Medico.* 1929. May 11. Vol. 43. No. 19. pp. 514–521. [Butantan Inst., Sao Paulo.]

The author points out the difference between mere "association" organisms in disease and "synergism" in which two organisms react beneficially or antagonistically upon each other. As examples he cites influenza bacillus and streptococci, hog-cholera and pasteurellosis, Proteus X19 and Rickettsia in typhus.

From the faeces of *M. rhesus* experimentally infected with yellow fever he isolated certain Gram-positive Corynebacteria which he designates M15, F3, F4 and F8. Of these he describes F4 ; it is a small bacillus 3 by 0·6 microns, pleomorphic and at times diplococcoid, fermenting dextrose and insulin slightly, laevulose strongly.

The sera from infected monkeys and from human cases from the third day of fever till convalescence agglutinated this in titres up to 1 in 320 and occasionally to 640. With typhoid, influenza and other diseases the results were negative and the author regards the reaction as of diagnostic value in early and doubtful cases of yellow fever.

H. Harold Scott.

DAVIS (N. C.) & BURKE (A. W.). **Studies on South American Yellow Fever. I. The Strains of Virus in Use at the Yellow Fever Laboratory in Bahia, Brazil.**—*Jl. Experim. Med.* 1929. June 1. Vol. 49. No. 6. pp. 975–984. [1 ref.] [Internat. Health Division, Rockefeller Foundation, New York.]

Five strains of yellow fever have been studied, three isolated from local cases of the disease, the other two obtained from Rio de Janeiro and Lagos respectively. The strain from Rio was not very virulent to monkeys, rarely causing death, whilst the one from Lagos, the Asibi strain, was highly virulent and nearly always fatal. Of the local strains the "B B" was isolated by the inoculation of blood collected from a fatal case 72½ hours after the patient was said to have been taken ill. The first monkey showed fever (104·3° F.) on the 11th day and transfer was made to another animal. This showed fever on the 7th day, the next two passages both on the 3rd day. Subsequently the virulence diminished, but was established again in mosquitoes. It has been carried through 14 passages and, out of 135 inoculated animals, 26 died or were sacrificed when moribund. The strain, however, is now much more fatal than these figures would indicate.

A second strain ("S.R.") was obtained from a very mild case of the disease, showing fever and albuminuria, but no jaundice or black vomit. Blood collected 66 hours after the onset was inoculated into a monkey, which showed fever after 7 days. Its blood was then inoculated into a second animal which died of yellow fever on the 5th day. Animals that recovered from this strain were immune to the B.B. virus, and also to the African virus.

A third strain ("J.V.O.") was isolated from a patient who died between 3 and 4 days after first showing signs of illness. Liver, spleen and kidney tissues collected at autopsy were inoculated separately into monkeys. That inoculated with spleen died of an intercurrent infection, the animal receiving liver remained unaffected and was later shown to be susceptible, as it died of yellow fever in 4 days when inoculated with the B.B. strain. The monkey that received kidney emulsion showed fever on various occasions but this was considered to be the result of a bacterial infection of the blood. On the 20th day the temperature rose to 105° F. and the monkey was killed. Sections of liver showed the presence of much fat but no necrosis. Liver and blood collected from this animal were inoculated into a second monkey whose temperature rose to 105·9° F. on the fourth day. On this day mosquitoes were fed on the monkey, which recovered from its infection. Later it was inoculated with a virulent strain of yellow fever (B.B.) and showed not the slightest reaction. The mosquitoes produced a slight and non-fatal infection in monkeys but although passages were made, the strain remained quite nonvirulent.

[The presence of virus in a monkey 20 days after the original inoculation is most unusual. Moreover, it is of interest that this animal had only been inoculated with kidney from a fatal case of the disease. This patient died after being ill for approximately only 80 hours, and this may explain why the virus had not yet disappeared, as hitherto the inoculation of material from human autopsies has given negative results.]

E. H.

DAVIS (Nelson C.). **Studies on South American Yellow Fever. II. Immunity of Recovered Monkeys to African Virus.**—*Jl. Experim. Med.* 1929. June 1. Vol. 49. No. 6. pp. 985–991. [3 refs.] [Internat. Health Division, Rockefeller Foundation, New York.]

Details are given of the results of inoculation of the Asibi strain of African virus into 22 monkeys recovered from Brazilian yellow fever, including four different strains. One of the monkeys succumbed to the African strain, although it had previously survived a typical attack of a Brazilian strain. The remaining monkeys were all immune. As the author remarks, although there are quantitative differences in virulence and minor differences in behaviour, the African and Brazilian strains of yellow fever virus that have been tested are immunologically the same. [See this *Bulletin, ante* p. 306.]

E. H.

DAVIS (Nelson C.) & SHANNON (Raymond C.). **Studies on South American Yellow Fever. III. Transmission of the Virus to Brazilian Monkeys: Preliminary Observations.**—*Jl. Experim. Med.* 1929. July 1. Vol. 50. No. 1. pp. 81–85. [2 refs.] [Internat. Health Div., Rockefeller Foundation, New York, & Yellow Fever Lab., Bahia, Brazil.]

A record of the transmission of yellow fever from rhesus monkeys to Brazilian monkeys, *Cebus macrocephalus*, both by blood inoculation and the bites of infected mosquitoes. The Cebus never developed any clinical or pathological signs of yellow fever, yet the virus persisted in the circulation for at least two days. Mosquitoes were infected by feeding on Cebus monkeys, and moreover, rhesus monkeys were infected by the inoculation of blood from Cebus that had either been injected with blood containing the virus, or bitten by infected mosquitoes.

E. H.

ARAGÃO (H. de Beaurepaire) & LIMA (A. da Costa). [In Portuguese & English.] Sobre a transmissão do virus da febre amarella pelas fezes de mosquitos infectados. [**On Transmission of the Virus of Yellow Fever by Excreta of Infected Mosquitoes.**]—*Inst. Oswaldo Cruz, Supplemento das Memorias.* 1929. June 22. No. 8. In Portuguese pp. 101–104. In English pp. 105–108. With 2 figs. on 1 plate; also in Portuguese in *Brasil-Medico.* 1929. June 15. Vol. 43. No. 24. pp. 669–671; and in French in *C.R. Soc. Biol.* 1929. Oct. 18. Vol. 102. No. 26. pp. 53–54. [Oswaldo Cruz Inst., Rio de Janeiro.]

—— & ——. [In Portuguese & English.] Sobre a infecção do *M. rhesus* pela deposição de fezes de mosquitos infectados sobre a pelle ou na conjunctiva ocular integras. [**On the Infection of** *M. rhesus* **by Contact of the Uninjured Skin and Conjunctiva with the Excreta of Infected Mosquitoes.**]—*Inst. Oswaldo Cruz, Supplemento das Memorias.* 1929. July 31. No. 9. In Portuguese pp. 133–135. [2 refs.] In English pp. 136–138. [2 refs.]

ARAGÃO (H. de Beaurepaire) & LIMA (A. da Costa). [In Portuguese & English.] Sobre o tempo necessario para que Stegomyias infectados excretem fezes virulentas. [**About the Time required by the Infected Stegomyia to expel Virulent Excreta.**]—*Ibid.* In Portuguese pp. 139–141. In English pp. 142–145.

——. [In Portuguese & English.] Infecção do *Aëdes aegypti* macho e possibilidade da propagação da febre amarella de Stegomyia a Stegomyia sem passagem pelo homem. [**On Infection of the Male "Aëdes aegypti" and the Possibility of Propagation of Yellow Fever from Stegomyia to Stegomyia without Passage through Man.**] —*Ibid.* In Portuguese pp. 190–192. In English pp. 193–195.

——. Possibilidade da infecção de *Aëdes aegypti* machos com virus da febre amarella. [**The Possibility of Infecting Male** *Aëdes aegypti* **with the Virus of Yellow Fever.**]—*Brasil-Medico.* 1929. June 15. Vol. 43. No. 24. p. 671; also in French in *C.R. Soc. Biol.* 1929. Oct. 18. Vol. 102. No. 26. pp. 54–56.

The authors collected the faeces of *Aëdes* sp. infected with yellow fever and inoculated them into *Macacus rhesus*. The results clearly show that the virus is present in the faeces of mosquitoes whose bites are infective. The virus in the faeces is able to produce infection through the ocular conjunctiva and also through the intact skin. In the latter case the authors carefully examined the surface of the skin with a binocular microscope before applying the virus, in order to be certain that no injuries were present. The virus was found to be present in the faeces of mosquitoes that had fed on an infected monkey respectively 5 and 7 days previously. The last of these papers, by Dr. Aragão, records the infection of male *Aëdes aegypti* by feeding them with a mixture of infected blood and honey. Three monkeys were infected by the inoculation of the contents of male mosquitoes respectively 13, 14 and 30 days after being fed on an emulsion containing the virus. The author then placed normal male mosquitoes in a cage with infected females and after 12 days the males were found to have become infected as tested by inoculating their contents into a monkey. The first monkey had a very mild infection, but a second monkey, inoculated with blood from the first, showed a typical attack of yellow fever. [Owing to a printer's error in the English account the number of the monkey is given as 425 instead of 426.]

Finally a monkey was inoculated with an emulsion of 4 female mosquitoes that had been left in contact with infected males for 17 days. This animal succumbed to a typical attack of yellow fever. These experiments show that in addition to the possibility of infecting male mosquitoes, female mosquitoes can become infected by being placed in contact with infected males, and *vice versa* male mosquitoes can acquire infection from contact with infected females. [These interesting experiments show that the virus can be spread among mosquitoes without the necessity of feeding on infected blood. It should be mentioned, however, that the positive results of the last experiments could be explained on the supposition that the insects had become soiled with the infective faeces of the opposite sex. It is difficult to conceive any manner in which the virus could pass through the chitinous covering of the male copulatory organs.]

E. H.

ARAGÃO (Henrique de Beaurepaire). Possibilidade da propagação directa de febre amarella de stegomyia a "Aëdes aegypti" sem intervenção do homem. [**The Possibility of Yellow Fever being transmitted directly by *Aëdes aegypti* without Human Intervention.**] —*Brasil-Medico.* 1929. Aug. 3. Vol. 43. No. 31. p. 885. [Oswaldo Cruz Inst., Rio de Janeiro.]

The author has recorded previously (see above) the infection of male Aëdes by allowing them to feed on defibrinated blood of an infected Rhesus, the mosquito being coaxed to feed by addition of 5 per cent. honey to the blood. He confirms these findings by two further similar tests, monkeys being infected with yellow fever by injection of an emulsion of male Aëdes which had so fed thirteen or more days before.

It is concluded, therefore, that: (1) Male *Aëdes aegypti* can be infected with the defibrinated blood of an infected *rhesus*; (2) Male Aëdes can become infected by being kept in contact with infected females and vice versâ; (3) Infection of mosquito by mosquito is possible, and, even though it may not be frequent, this fact may serve to explain the occurrence of yellow fever at long intervals without detection of intermediate cases.

H. Harold Scott.

CHAGAS (Carlos). La fièvre jaune. Recherches expérimentales effectuées à l'Institut Oswaldo Cruz. [**Yellow Fever. Experiments performed at the Oswaldo Cruz Institute.**]—*Bull. Soc. Path. Exot.* 1929. June 12. Vol. 22. No. 6. pp. 398–407.

A summary of recent work on yellow fever at the Oswaldo Cruz Institute. The only new information is contained in a brief reference to the first results of using yellow fever vaccine. During this year's epidemic in Rio more than 15,000 persons were vaccinated, the subjects being mainly immigrants or persons particularly exposed to the chance of infection, and 14 became infected. The author considers that the dosage was probably insufficient to overcome the extreme susceptibility of certain individuals. [Aragão's modification of Hindle's vaccine was used in these experiments. See this *Bulletin, ante,* p. 303–4.]

E. H.

VELLARD (J.) & VIANNA (M.). Modifications de la coagulation sanguine dans la fièvre jaune; leur importance pour le diagnostic précoce. [**Modifications in the Coagulation of the Blood in Yellow Fever, their Importance for Early Diagnosis.**]—*C.R. Acad. Sci.* 1929. May 27. Vol. 188. No. 22. pp. 1452–1455. [1 ref.]

—— & ——. Modifications de la coagulation sanguine dans la fièvre jaune.—*Inst. Oswaldo Cruz, Supplemento das Memorias.* 1929. May. No. 7. pp. 86–99. With 3 plates. [Vital Brazil Inst., Nictheroy.]

—— & ——. Modifications de la coagulation du sang dans la fièvre jaune.—*C.R. Soc. Biol.* 1929. July 17. Vol. 101. No. 24. pp. 961–962. [1 ref.]

——. Modificações da coagulação sanguinea na febre amarella.—*Brasil-Medico.* 1929. May 25. Vol. 43. No. 21. pp. 588–596. With 2 charts.

The diminution in the coagulability of the blood of yellow fever cases has often been recorded, without its origin having been determined. The authors, therefore, made a careful study of the blood in 25 cases

of yellow fever, 12 convalescents and 7 doubtful cases. The results indicate that the coagulating properties of the serum are very irregular resembling those of normal individuals. The coagulability of the plasma, from the second day of the disease, is invariably very much diminished, reaching its greatest diminution on the 7th to the 9th days, after which it gradually rises again. The changes are mainly due to the appearance of large quantities of antithrombin in the blood, there being little change in the amount of fibrinogen. The diminution in the coagulability exceeds that in any other disease studied by the authors, and may be of use in the early diagnosis of yellow fever. From the point of view of prognosis, a rapid and extreme diminution is a very grave sign, as it indicates profound changes in the liver.

*Macacus rhesus*, infected with yellow fever, showed similar changes in the blood, but these seemed to develop more slowly and were less evident than in human cases.

E. H.

VELLARD (J.) & VIANNA (Miguelotte). Modifications de la coagulation sanguine au cours de la fièvre jaune expérimentale chez le *Macacus rhesus*. [**Modifications in the Blood Coagulation during the Course of Experimental Yellow Fever in** *Macacus rhesus*.]—*C.R. Acad. Sci.* 1929. Sept. 16. Vol. 189. No. 12. pp. 430–432.

The authors have studied this property in 16 monkeys infected with the Brazilian virus and obtained results resembling, but to a lesser degree, those obtained in human beings [see above]. The anti-coagulating power is never complete, which is in accordance with the circumscribed haemorrhages observed in monkeys. The animals which recovered from the disease showed no diminution in the coagulability of the blood.

E. H.

CRUZ (J. da Costa). Teneur du sérum en alexine dans la fièvre jaune. [**The Amount of Complement in Yellow Fever Serum.**]—*C.R. Soc. Biol.* 1929. July 17. Vol. 101. No. 24. pp. 948–949.

——. Diagnostic de la fièvre jaune par le dosage de l'alexine.—*Ibid.* pp. 954–956. [1 ref.] [Oswaldo Cruz Inst., Rio de Janeiro.]

The author has tested the amount of complement present in the sera of eight yellow fever patients and six convalescents. The results indicate a marked diminution to less than a quarter in the blood of yellow fever patients, due to a real absence of complement and not to the development of anticomplement in the sera. During convalescence the amount of complement was found to become rapidly re-established, and in two cases was normal only four days after the fever had disappeared. In two other cases, 15 days after recovery, the amount was in excess of normal. The author remarks that in acute infectious diseases, it is very rare to observe any diminution in the amount of complement, and consequently this test may be of help in the diagnosis of doubtful cases.

In the second article further evidence is adduced in support of the value of this test. 103 patients were examined of whom 51 showed lack of complement. Of these 51, 46 were diagnosed clinically as yellow fever and in only one instance was the complement reaction negative and the clinical diagnosis positive for this disease.

E. H.

FROBISHER, Jr. (Martin). **The Complement Fixation Test in Yellow Fever.**—*Proc. Soc. Experim. Biol. & Med.* 1929. June. Vol. 26. No. 9. pp. 846–848. [4 refs.] [Internat. Health Division, Rockefeller Foundation, New York.]

After trying several antigens the author found that the most satisfactory results were obtained with a saline extract of liver and spleen. Hindle's method [*ante*, p. 292] was employed for extracting the virus from the tissues. whereby the cells are exposed to sudden changes in osmotic pressure, and the resulting suspension was centrifuged and passed through Berkefeld V filters. The filtrate constituted the antigen. As control, an antigen was similarly prepared from the tissues of a normal monkey. The results of complement fixation tests are given in the following table :—

RESULT OF COMPLEMENT FIXATION TESTS IN YELLOW FEVER.

| Sources of sera tested. | Antigen Used. | | | | | |
|---|---|---|---|---|---|---|
| | Saline extract of liver and spleen of yellow fever monkeys. | | | Saline extract of liver and spleen of normal monkeys. | | |
| | Positive. | Negative. | % Correct. | Positive. | Negative. | % Correct. |
| Normal men and monkeys ... ... | 3 | 38 | 93 | 1 | 28 | 97 |
| Recovered and immune men and monkeys ... | 18 | 5 | 78 | 3 | 9 | 75 |
| Convalescents from other diseases* ... | 1† | 19 | 95 | 0 | 18 | 100 |

* Syphilis 14, dengue 4, poliomyelitis 2 (1 human, 1 monkey).
† Syphilis.

The results clearly indicate that this method offers a means of identifying a large percentage of yellow fever convalescents. Sera collected on the first day of fever did not give strong or frequent reactions and the presence of syphilis antigens interfered with the fixation of complement in yellow fever convalescents. Although the antigen was prepared from a monkey infected with a West African strain of the disease, positive reactions were obtained with the sera of both monkeys and human beings infected with the Brazilian strain.

E. H.

MOSES (Arthur). Reacções sorologicas na febre amarella. [**Serum Reactions in Yellow Fever.**]—*Archivos de Hyg.* Rio de Janeiro. 1929. Vol. 3. No. 1. pp. 27–35. [10 refs.] French summary facing p. 35.

By using " cocto-antigens " containing yellow fever virus, and using exact methods of estimating the results of haemolysis and precipitation, both anti-bodies and precipitins were shown to be present in the serum of yellow fever patients. The antibodies were present from the 3rd to the 24th day of the disease, but especially on the 5th and 6th

days, after which they gradually disappeared. They were not found in the sera of patients suffering from other diseases, and the reactions were only obtained with the specific antigens. In the serum of rabbits hyperimmunized by intravenous inoculations of the yellow fever material used as antigen, the same antibodies were found.

E. H.

HUDSON (N. Paul), PHILIP (Cornelius B.) & DAVIS (Gordon E.). **Protection Tests with Serum of Persons recovered from Yellow Fever in the Western Hemisphere and West Africa.**—*Amer. Jl. Trop. Med.* 1929. July. Vol. 9. No. 4. pp. 223-232. [3 refs.]

The sera from five out of six persons convalescent from yellow fever in Brazil, in doses of 1 cc. and 2 cc., protected monkeys against the West African virus. The serum of one convalescent failed to protect against the inoculation of virus, but subsequently a monkey inoculated with 1 cc. of the same serum, was bitten by 5 infected mosquitoes without showing any signs of infection.

The sera of four natives who had yellow fever in West Africa were also tested, by the simultaneous inoculation of serum and virus into monkeys. Three of the sera were protective, and the one that failed protected 6 monkeys in other tests before and after the one in question. These results furnish additional evidence of the identity of yellow fever in America and West Africa. [See above.]

E. H.

PHILIP (Cornelius B.). **Preliminary Report of Further Tests with Yellow Fever Transmission by Mosquitoes other than** *Aëdes aegypti*.—*Amer. Jl. Trop. Med.* 1929. July. Vol. 9. No. 4. pp. 267–269. [3 refs.] [Internat. Health Division of Rockefeller Foundation, Lagos.]

Three more species have been shown to be capable of transmitting yellow fever to monkeys by their bites, in addition to those recorded by BAUER [this *Bulletin*, Vol. 25, p. 848]. These are *Aëdes vittatus* (Bigot) (=*sugens* Theo.) a common semi-domestic species; *Aëdes africanus* (Theo.), a 'bush' species which is partial to bamboo stumps; *Aëdes simpsoni* (Theo.), an infrequent tree-hole breeder in Lagos, but more common inland. This latter species is a particularly willing biter in the laboratory, and will feed on human beings as readily as *A. aegypti*.

[This brings the list of mosquitoes shown to be capable of transmitting the disease experimentally up to a total of seven, and indicates that the epidemiology of yellow fever is not such a simple matter as was previously supposed.]

E. H.

MARCHOUX (E.). La fièvre jaune et la sensibilité du *Macacus rhesus*. [**Yellow Fever and the Susceptibility of** *Macacus rhesus*.]—*Ann. Inst. Pasteur.* 1929. June. Vol. 43. No. 6. pp. 737–748. [18 refs.]

The author again calls attention to the fact that the experiences of the French Yellow Fever Commission in Brazil (1903) were against the view that the virus of this disease is able to pass through the skin. In support of this view it is mentioned that the members of the Commission

performed about one hundred autopsies with bare hands, yet none of them became infected. [Recent experiments, however, have shown that in human cases the virus practically always disappears from the blood and organs before death.] Also their hands were often soiled with blood when collecting blood from the veins of patients.

In order to find out if the virus could pass through the unbroken epidermis a few experiments were made. Blood was collected from a monkey two days after inoculation. After defibrinating, some of the blood was inoculated subcutaneously into a monkey which showed no signs of infection, but was immunized against a subsequent inoculation of infected liver. A second monkey had some of the blood placed on excoriated skin, and allowed to dry. It remained uninfected but when the procedure was repeated with an emulsion of infected liver it became infected and died of yellow fever. A third monkey had the same quantity of blood placed on the *intact* skin, and later, infected liver material, but remained healthy. A fourth monkey received a drop of the defibrinated blood in the conjunctival sac of the right eye and remained uninfected; when subsequently infected liver emulsion was similarly introduced, the animal died of yellow fever five days later. A control animal inoculated intraperitoneally with the liver emulsion died of the disease three days later. Infected liver preserved in the ice chest was found to have lost its virulence and also its power of conferring immunity within five months. The author discusses prophylactic measures and points out the necessity of maintaining anti-mosquito measures in all endemic regions. Usually, these are relaxed after a few years and conditions are then liable to favour the development of fresh outbreaks of the disease. Finally, it is stated that in both Africa and America where the mean isotherm is above 20° C., a country is not safe from yellow fever unless the number of mosquitoes is less than 10 units for each area, and the number of *Aëdes* less than 2 per cent. of the total.

E. H.

DINGER (J. E.), SCHÜFFNER (W. A. P.), SNIJDERS (E. P.) & SWELLENGREBEL (N. H.). Onderzoek over gele koorts in Nederland. (Eerste mededeeling). [**Yellow Fever Research in Holland. (First Communication).**]—*Nederl. Tijdschr. v. Geneesk.* 1929. July 13. Year 73. 2nd Half. No. 28. pp. 3255–3257.

Since *Macacus rhesus* has been found to be susceptible to yellow fever new paths for research are opened, and the authors took up the question of the possibility of transmission of yellow fever under natural circumstances in the Dutch East Indies. In view of the danger of such experiments in the tropics, they were carried out at Amsterdam with monkeys (*Macacus cynomolgus*) and mosquitoes (*Aëdes aegypti*) from Java. The virus was obtained from Prof. PETTIT (Paris) in the shape of frozen blood and liver of an infected monkey. It proved to be highly virulent. Of 6 infected cynomolgus monkeys one died. It did not show the typical post-mortem picture, but the virus was shown to be present in its organs by successful inoculation into two rhesus monkeys. Possibly the 5 other cynomolgi had a light type of the disease. Anyhow, the cynomolgi are to be considered as a possible reservoir of the virus.

The bite of *Aëdes aegypti*, 19 days after its sucking the blood of an infected monkey, was fatal to a healthy rhesus. Accordingly, the

mosquito is susceptible to the yellow fever virus. A natural immunity of the D.E.I. does not exist. In view of the possibility of import of the disease from America, this conclusion is of great practical importance.

The experiments are to be continued. W. J. Bais.

SAWYER (W. A.), LLOYD (W. D. M.) & KITCHEN (S. F.). **The Preservation of Yellow Fever Virus.**—*Jl. Experim. Med.* 1929. July 1. Vol. 50. No. 1. pp. 1–13. [8 refs.] [Internat. Health Div., Rockefeller Foundation, & Rockefeller Inst. for Med. Research, New York.]

A detailed account of experiments on various methods of preserving yellow fever virus. As noted by previous observers the virus may be preserved if dried in vacuo. The best method was found to be freezing and drying blood collected from a monkey on the first day of an attack of the disease, and storing the dry material in sealed glass tubes containing calcium chloride. The dried virus when kept in a refrigerator has been preserved for at least 154 days. The virus may be preserved for at least 30 days in liver kept frozen but clotted or citrated blood soon loses its virulence. Infected blood or liver, in 50 per cent. glycerine stored in the refrigerator, usually contains virus for at least 60 days and possibly 100 days but the older material gradually loses its virulence.

[No reference is made to the reviewer's experiments with dried virus. This method of preservation was first recorded in July, 1928 (this *Bulletin*, Vol. 25, p. 850), and details of experiments on this and other methods of preservation were published in January, 1929, *ante*, p. 292.]

E. H.

PETTIT (Auguste), STEFANOPOULO (Georges) & KOLOCHINE (Constantin). Conservation du virus amaril. [**The Preservation of Yellow Fever Virus.**]—*Bull. Acad. Méd.* 1929. July 30. Year 93. 3rd Ser. Vol. 102. No. 29. pp. 98–104. [7 refs.]

A useful summary of recent observations on this subject in which the author includes his own experiences. The best method of preserving infected liver or blood in the refrigerator was to mix it with a glycerine phosphate mixture recommended by VALLÉ. This consists of equal parts of 0·235 per cent. dipotassium phosphate, and 0·090 per cent. monopotassium phosphate. These are mixed and glycerine added equal to the quantity of the phosphate mixture. Intraperitoneal and intrahepatic inoculations were found to be more certain than the subcutaneous method. The author has seen monkeys die of yellow fever without showing any febrile symptom. Jaundice was never clearly developed with the Senegal strain, but a monkey infected with the Asibi strain showed very evident jaundice. The duration of the disease in the case of monkeys infected with the Senegal strain, has varied between 3 and 8 days and the average duration for 14 months is given in tabular form. The results start with an average of 4·6 and finish with 5·2, so there has been no appreciable variation during this period. The Asibi strain has given comparable results, the first monkey dying in 5 days. Two *Macacus rhesus* and one *M. cynomolgus*, which were resistant to the Senegal virus, were also resistant to the Asibi strain.

E. H.

HOFFMANN (W. H.). Europa und das Gelbfieber. [**Europe and Yellow Fever.**]—*Muench. Med. Woch.* 1929. Sept. 27. Vol. 76. No. 39. pp. 1623–1627. [Finlay Inst., Havana, Cuba.]

A general discussion of the present position of the epidemiology of yellow fever, calling attention to the fact that West Africa is a potential source of danger to Europe.

E. H.

BARRETO (João de Barros). Notas epidemiologicas sobre a febre amarella no Rio de Janeiro, em 1928. [**Epidemiological Data on the Yellow Fever Outbreak in Rio (1928).**]—*Archivos de Hyg.* Rio de Janeiro. 1929. Vol. 3. No. 1. pp. 93–193. With 15 figs. on 6 plates & 1 folding map. English summary.

A detailed account of this outbreak with epidemiological observations on 175 individual cases of yellow fever.

E. H.

PETTIT (Auguste). A propos de la fièvre jaune. [**Concerning Yellow Fever.**]—*Rev. Méd.-Chirurg. des Maladies du Foie.* Paris. 1929. Mar.–Apr. Vol. 4. No. 2. pp. 133–139. [1 ref.]

A summary of recent work on the subject. Nothing new.

E. H.

ARAGÃO (Henrique de Beaurepaire). Febre amarella experimental do Brasil. [**Experimental Yellow Fever in Brazil.**]—*Brasil-Medico.* 1929. July 27. Vol. 43. No. 30. pp. 849–855.

This paper, read before the South American Hygiene Conference, gives an account of the work of the author and others on yellow fever—the search for the virus, the hypothetically aetiological 'germs,' Cryptococcus xanthogenicus, Leptospira icteroides, B. hepatodystrophicans, etc., the serological diagnosis and the preparation and use of the vaccine—but contains nothing which has not already received notice in the *Bulletin.*

H. Harold Scott.

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BARBOSA (J. Placido). Pequena historia da febre amarella no Brasil.—*Archivos de Hyg.* Rio de Janeiro. 1929. Vol. 3. No. 1. pp. 5–25. [9 refs.] English summary.

BARROSO (Sebastião). O problema da febre amarella no Brasil—a liçao dos factos.—*Brasil-Medico.* 1929. Apr. 27. Vol. 43. No. 17. pp. 455–457.

LINTZ (Alcides) & PARREIRAS (Decio). Aspectos epidemiologicos e prophylacticos da campanha de febre amarella no Estado do Rio de Janeiro em 1928.—*Folha Med.* 1929. Aug. 15. Vol. 10. No. 23. pp. 280–287.

OTERO (Francisco). Fiebre amarilla. Un caso da forma adinámica, atendido en el hospital flotante "Doctor Rodolfo del Viso."—*Semana Méd.* 1929. May 16. Vol. 36. No. 20 (1844). pp. 1264–1267.

OTERO (Francisco). Fiebre amarilla. Su diagnóstico diferencial.—*Semana Méd.* 1929. June 27. Vol. 36. No. 26 (1850). pp. 1682–1685. [2 refs.]

OTERO (Francisco). Fiebre amarilla. Su anatomia patológica.—*Semana Méd.* 1929. July 25. Vol. 36. No. 30 (1854). pp. 261–265. [1 ref.]

PENNA (Oswino) & DE FIGUEIREDO (Burle). Contribuição ao estudo da histopathologia do figado na febre amarella.—*Folha Med.* 1929. July 15. Vol. 10. No. 20. pp. 229–233. With 6 text figs.

REGENDANZ (P.). Resumo des resultados das pesquizas sobre a febre amarella.—Reprinted from *Revista Méd.* 1929. No. 7. pp. 467–473. [46 refs.]

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## BLACKWATER FEVER.

RUGE (Reinhold). Ein Beitrag zur Schwarzwasserfieber-Frage. [**The Blackwater Fever Question.**]—*Arch. f. Schiffs- u. Trop.-Hyg.* 1929. June. Vol. 33. No. 6. pp. 328–330. [6 refs.]

Of the many hypotheses regarding the etiology of blackwater fever that of NOCHT seems to have most to support it. NOCHT believes that in all three species of malaria parasites particular strains occur which exhibit a special haemolytic action just as is the case with certain strains of bacteria. The author considers that the following facts support this view :—

1. Most cases of blackwater fever occur in tropical infections: quotations from various authors are given in support of this statement.

2. H. RUGE (1928) found that amongst the cases of malaria coming from West Africa and treated at Hamburg, about 96 per cent. were *Tropica*; 4·2 per cent. developed blackwater fever and of these only 0·43 per cent. came from other parts of the world. The West African *Tropica* alone had a blackwater morbidity of 4·4 per cent., and the *Tropica* of other parts of the World nil. Even in the unusually severe intractable Brazilian malaria (*Tropica*+*Tertian*) in which the number of relapses was 25 times as high as in other forms, the blackwater fever morbidity was only 1·2 per cent.

3. This suggests that the West African malaria has a particular haemolytic action, and the question arises whether the West African *Tropica* parasite exhibits any differences— morphological or biological —from that found in other parts of the world.

4. The dispute concerning the unity of the *Tropica* parasites is not yet settled; some authors distinguish three species amongst them and others two. Ruge agrees with ZIEMANN. He believes that he can distinguish between the parasite from West Africa and that from other parts of the world. The schizont of the West African form is, as a rule, smaller than that of the other forms and, secondly, very rarely grows to larger rings in the peripheral blood, so that only small and very small rings are to be seen. He also agrees with ZIEMANN with regard to specificity of the crescents and the rareness with which they are met with in preparations of blood from West Africa.

Ruge considers it is this particular West African parasite, which is more rarely found in other parts of the world, that is specially concerned with the haemolysis in blackwater fever.

W. Yorke.

COLLIGNON (M.). Les hémoglobinuries. [**The Haemoglobinurias.**]—*Arch. Méd. et Pharm. Milit.* 1929. May. Vol. 90. No. 5. pp. 615–641. [13 refs.]

The article commences with a description of various methods of diagnosing haemoglobinuria and a list of the conditions with which it could be confused. The author classifies the haemoglobinurias as follows :—

1. *Experimental haemoglobinurias.* These can be easily produced by an intravenous injection of a sufficient quantity of haemoglobin.

Haemoglobin exists both in the blood and in the striated muscle, and the venous injection of either results in haemoglobinuria.

2. *Clinical haemoglobinurias.* He classifies these as follows :—

### *A. Haemoglobinurias without icterus.*

(*a*) *Toxic haemoglobinurias.*—These are most closely related to the experimental haemoglobinurias ; they may be the result of drug intoxication, alimentary intoxication, the introduction into the organism through the body or injection of organic haemolytic substances, such as venom or haemolytic sera, and, finally, the resorption of certain organic liquids, such as that resulting from the contortion of the pedicle of an ovarian cyst.

(*b*) *Infectious haemoglobinurias.*—These may be met with in the course of septicaemia, after typhoid, pneumonia, scarlatina, diphtheria, tetanus, etc. Into this group fall the haemoglobinurias due to piroplasma.

(*c*) *Paroxysmal haemoglobinuria.*—A summary of the clinical signs and of the complications of this condition is given and the predisposing causes are discussed.

### *B. Haemoglobinurias with icterus.*

(*a*) *Bilious haemoglobinuric fever.*—A brief description of blackwater is given which contains nothing new.

(*b*) *Other haemoglobinurias.*—Under this heading reference is made to a disease described by WIDAL due to the *Bacillus perfringens* which, when inoculated into animals, gives rise to a very marked haemolysis.

There is also a group of cases of combined icterus and haemoglobinuria of unknown cause, as, for example, the case published by CHAUFFARD and VINCENT. Such cases seem to be allied to the toxic haemoglobinurias. Finally, there are the cases of simple acholuric jaundice with intermittent haemoglobinuria.

When one is confronted with a case of haemoglobinuria, one must remember that there are five possible origins of the haemoglobinuria.

1. *Urinary origin.*—This is very rare ; VAN ROOSEN believes that lysis of the red cells in the bladder may take place in the presence of oxalates in excess, but it is doubtful whether this view is correct. There probably exist, however, some cases of haematuria in which the red cells undergo haemolysis in the urine, thus giving rise to a false haemoglobinuria.

2. *Renal origin.*—This origin is considered by certain authors to explain a considerable number of haemoglobinurias. It is believed that the site of the haemolysis is the renal parenchyma. It is seen in patients who are suffering from nephritis with intrarenal haemorrhages, or in patients in whom exposure to cold provokes a peripheral constriction resulting in an intense renal congestion with small haemorrhages. In these cases haemolysis takes place of the red cells which have escaped from the vessels, and the haemoglobin escapes in the urine.

3. *Muscular origin.*—This is evidently very rare in human pathology. In such cases the myo-haemoglobin is eliminated by the kidneys. This theory has its origin in facts obtained from veterinary medicine. In the horse, for example, paroxysmal haemoglobinuria of muscular origin is a well-known disease.

4. *Blood origin.*—Such cases may be due to alterations in the serum or alterations in the red cells. To the former belong paroxysmal

haemoglobinuria or *haemoglobinuria a frigore.* In some cases, it is possible to correlate haemoglobinuria with alterations in the red blood cells. Examples are quoted where the fragility of the red cells to hypertonic solutions and normal sera is altered.

5. *A complex, splenic, hepatic, blood, and renal origin.*—The author briefly discusses the various theories relating to this subject.

W. Y.

i. STEPHENS (J. W. W.). **The Distribution of Blackwater Fever in India.**—*Ann. Trop. Med. & Parasit.* 1928. Aug. 28. Vol. 22. No. 2. pp. 169–178. With 2 maps. [36 refs.]

——. **The Distribution of Blackwater Fever in Burma and the Far East.**—*Ibid.* pp. 179–199. With 4 maps. [76 refs.]

ii. ——. **The Distribution of Blackwater Fever in Africa.**—*Ibid.* 1929. Apr. 26. Vol. 23. No. 1. pp. 67–102. With 2 maps. [133 refs.]

i. These two articles consist entirely of tables showing the distribution of blackwater fever in India and in Burma and the Far East, respectively.

ii. The distribution of blackwater in Africa is given in tabular form. It is impossible to summarize the work, and it should be consulted in the original by those interested.

W. Y.

WHITMORE (Eugene R.) **Malarial Hemoglobinuria.**—*Sixteenth Ann. Rep. Med. Dept. United Fruit Company, Boston, Mass.* 1927. pp. 101–113. With 2 text figs. [4 refs.]

——. **Blackwater Fever.**—*Ann. Internal Med.* 1928. Oct. Vol. 2. No. 4. pp. 316–324. With 7 charts on folding sheet. [Georgetown Univ., Washington, D.C.]

The author has again turned his attention to malarial haemoglobinuria and, through the courtesy of Dr. DEEKS and others, was enabled to study the disease in the Banes and Preston divisions (Cuba) of the United Fruit Company, and to visit Jamaica and Panama for the purpose of collecting data. The work followed two lines: (*a*) Epidemiological Studies, and (*b*) Laboratory Investigations. Only the epidemiological studies are referred to in the present paper.

The author first deals with the question of susceptibility, with a view to obtaining an explanation of the occurrence of haemoglobinuria in some cases of malaria, but not in all. As regards racial susceptibility, there is general agreement that some races are more susceptible to blackwater than others, and this is usually explained as the result of the development of a relative immunity to malaria after prolonged residence—probably generations—in the malarious region. In a table, the malaria and blackwater rates for American, European, and Negro employees in the Canal Zone from 1905 to 1910 are given. Both rates are much higher in the European than in the Negro employees; the low rates for the American employees were in a great part due to better living conditions, and also to the high labour turnover

amongst them. From a consideration of the annual reports of the Canal Commission, it seems clear that the Negro labour is extensively exposed to malarial infection, and that the living conditions and nutrition of this class were not good; yet malaria and blackwater fever rates were much lower in it than in the European class. There is no evidence of difference of susceptibility among the Europeans. In Jamaica, blackwater fever was rare; no reports were obtained in the case of negroes.

The conditions in Cuba enabled the author to study blackwater in a white race which has lived for generations in the region. The Cuban race is mainly Spanish. The blood of the original Indian races probably played a relatively small part in the formation of the Cuban race, and the blood of the African slaves has become amalgamated with that of the lower and middle classes of the Cuban race. Blackwater fever is rare among the Haitians, who were imported for the cane cutting season, but during four months of the year it is fairly common among the Cubans in the same area. The disease is very rare among the Jamaican negroes living in Cuba.

The author next turns to the question of family susceptibility. Some interesting information was obtained, and the data concerning a few representative families, several members of which had suffered from blackwater fever, are given. This is followed by information concerning two families, no member of which had suffered from blackwater, notwithstanding the fact that they had presumably all suffered from malaria.

Dealing with the subject of individual susceptibility, the author observes that it has long been recognized that the person who has had one attack of blackwater is quite liable to have another attack. Among persons coming from a non-malarious region, the disease occurs most frequently within 12 to 18 months after beginning residence in the malarious district. The statement that long residence of an individual reduces the liability to blackwater is usually explained as due to an acquired immunity to malaria, and so to blackwater; but the conditions in Cuba, with a population born and spending its life in the malarious region, do not substantiate this idea. Blackwater is common in Cuban children, and is very likely to develop before the person is twenty years of age; this is undoubtedly associated with the early opportunity for the development of chronic malaria. Some of the children, however, lived in a malarious region until they were 16 or 18 before developing blackwater. There was here as elsewhere a tendency for a person who had had one attack of blackwater to develop another. The author asks whether it is possible that we are dealing with an individual susceptibility, and that the susceptible persons develop blackwater after a shorter period of chronic malaria than the less susceptible person, whilst the non-susceptible do not develop it at all.

The next section relates to quinine and blackwater fever. No one can doubt the possibility that quinine precipitates a case of blackwater. Everyone who has worked with the condition has seen cases in which he is satisfied that quinine precipitated the attack, though usually it is impossible to say whether or not the attack might have occurred in the absence of quinine. Cold will also precipitate an attack, and possibly also fatigue. All who have worked with malaria are assured that malaria is the primary factor, and that, except in persons who are especially susceptible to quinine, this drug is simply the last straw in the

load. Many cases have been seen in which malaria was the only factor in persons never having taken quinine. Such a case came under the notice of the author in Cuba.

The author closes his article with the following sentence :—

" Everything supports the belief that the efficient and intelligent administration of quinine in the treatment of malaria, as well as its utilization when required in malaria prophylaxis, along with other methods of malaria prophylaxis, does not increase the incidence of blackwater fever, but that in fact it markedly decreases the incidence of blackwater fever."

W. Y.

MENK (Walter). **Notes on Blackwater Fever in Banes Hospital, Banes, Cuba.**—*Sixteenth Ann. Rep. Med. Dept. United Fruit Company, Boston, Mass.* 1927. pp. 113–117. [2 refs.]

Blackwater fever in the Banes Division shows epidemiologically the same features as have been described in the Panama Canal Zone. The disease exhibits a marked tendency towards the same seasonal incidence as malaria, the apex in the incidence of blackwater being slightly behind that of malaria. *P. falciparum* was found in blood films of about 50 per cent. of the patients ; the other two malaria parasites were not found. Parasites were more commonly found in those suffering from the initial attack of blackwater than in the second or third attack. No piroplasma, spirochaete, or other type of organism was found. The disease was seldom observed in a large negro population, although they were more exposed to malarial infection than the rest of the population and their parasitic rate was very high. Blackwater fever, as a rule, occurs in the Cubans of predominently white extraction ; in Spaniards who have lived in unscreened quarters ; and in individuals who neglect treatment for malaria.

A number of clinical observations are discussed. These relate to (1) Race incidence, (2) Number of attacks in the cases, (3) Age incidence, (4) Intervals between paroxysms of haemoglobinuria, (5) Duration of paroxysms of haemoglobinuria, (6) Mortality rates, (7) Duration of stay in hospital, and (8) Treatment.

Menk states that there is a rapid reduction in the total number of the red cells during the haemoglobinuric paroxysms, and in severe cases the count may fall to 1,000,000 in a few days. He adds that " the red-cell count gives a more accurate idea of the progress of the anaemia than the haemoglobin estimation (Tallquist), which may offer false values when made during the period while the blood-serum is deeply coloured."

[This seems to the reviewer to be rather a loose statement. It implies that the blood serum in this disease contains a very considerable amount of haemoglobin. The statement that the blood serum is deeply coloured appears from time to time in the literature of blackwater fever, but such quantitative estimations of the degree of haemoglobinaemia as have been made have not revealed a higher concentration than that contained in about a 1 per cent. solution of normal red cells.]

Marked basophilia, polychromatophilia, anisocytosis, and poikilocytosis may be seen. A marked difference was observed in the staining properties exhibited by many of the red cells. Achromia and ghost cells are sometimes seen, as were also *Corps en demilune* and Cabot's

rings. The nucleated red cells were not found earlier than the 4th or 5th days of the disease. In the first stage of the disease the leucocyte count was usually normal, but later there was a tendency to leucocytosis, whilst in the convalescent period leucocytes may fall as low as 4,000 or 5,000. Even at the onset of the attack, a high percentage of immature neutrophiles appear, and in severe and fatal cases they may reach an astonishing number.

The appearance of the blood serum taken at the height of the attack is so characteristic, the author states, that he could not imagine anything being the cause of it other than haemoglobinaemia. The test for haemoglobin was very strongly positive in several of these cases, a positive reaction in 1 in 1,000 dilution of one dark red and one dark brown specimen of blood serum being obtained. The author estimates that haemoglobin content of the serum in blackwater is about 25 to 30 times higher than in that of the controls. The sedimentation time of the red cells was determined by the Linsemeyer technique and was found to be decidedly decreased. The complement content of the blood serum was examined in three cases, but no definite conclusion could be drawn, except that the duration of the intervals at the end of the haemoglobinuria period are not primarily determined by the disappearance of complement.

W. Y.

CORMACK (R. P.). **Report on Blackwater Fever in Uganda for 1927.**—*Uganda Protectorate Ann. Med. & San. Rep. for Year ended 31st December, 1927.* Appendix No. III. pp. 75–78. With 1 chart.

During the year 1927, 106 cases of blackwater fever with 28 deaths (26·4 per cent.) occurred in Uganda. In a table the number of cases and deaths reported during each of the last 20 years is given. From this table it appears that during this period 1,245 cases of the disease had been recorded. The number of deaths totalled 296 (23·8 per cent.). Analysis of the figures obtained during the last six years shows that there are three periods of greatest intensity of the disease. The most important peak is in July, and the others are in March, and October or November. In another table, the incidence of the disease during the present year amongst officials, non-officials, race and sex is shown. In Buganda the morbidity rate was greater among Asiatics than among Europeans, whilst in the Eastern Province the reverse was the case. Regarding the periods of residence in the tropics before the attack, the following information was obtained. One case had been less than one year in residence, 4 cases less than 2 years, 7 cases less than 5 years, 10 cases less than 10 years, 14 less than 20 years, and 16 less than 25 years; all these were Europeans. Only one of the European cases is reported to have suffered from the disease previously. It was that of a woman with about 23 years residence in the tropics, who had had two previous attacks in 1914. In 103 of 106 cases there were previous attacks of malaria. A low standard of living and housing, and the liability to frequent bites from mosquitoes, combined with repeated and recent attacks of malaria inadequately treated, appeared to be potent predisposing causes. Chill, fatigue, quinine, and recent malaria are stated to be the exciting causes. The authors omitted to classify the cases according to whether or not the patients

were in the habit of taking quinine prophylactically. It is stated that 11 took quinine regularly, 27 took it irregularly, 14 did not take it, and in 3 no information was available. Information is given regarding the state of the blood before and during the attack in a number of cases.

W. Y.

West African Medical Journal. 1929. Jan. Vol. 2. No. 3. pp. 164–171.—**Monthly Meeting of the Medical Practitioners of Lagos. Discussion on Blackwater Fever** [Thomson (J. W.); Gray (G. M.); Connal (Andrew); Wakeman (A. M.)].

Dr. Thomson, who opened the discussion, gave details regarding several cases of blackwater, and emphasized the importance in treatment of administering large quantities of fluid. He then passed on to a consideration of factors which might be concerned in the production of blackwater. Dr. Connal quoted a number of figures taken from reports sent in by medical officers during the past 13 years. Between 1915 and 1927, he received reports of 291 cases of which 157 came from Northern Provinces and 134 from the Southern Provinces. It should be noted that there are many more Europeans in the Southern Provinces than there are in the Northern Provinces. The months in which the cases occur are shown in a table. Information is also given regarding the length of residence in malaria infected countries and of the quinine habits of the patients.

Drs. Gray and Wakeman gave a detailed account of a case which recovered after nearly five days of complete anuria.

The patient felt seedy for a week before entering hospital, and three days before admission he had severe chills and had taken 20 grains of quinine, and 10 grs. on each of the two following days. At 3 a.m. on September 29th, 1928, the date of admission to hospital, he had a very severe chill, uncontrollable vomiting, quickly became prostrate and finally collapsed unconscious. There was no history of any haemoglobinuria before admission. On arrival in hospital at 6 a.m., the patient had regained consciousness and complained of nausea, thirst, and severe abdominal pain. The first urine passed about 10 a.m. was black and measured less than 2 ozs. During the next 24 hours he voided less than 1 oz. of similar urine and then developed complete anuria which lasted for five days. On October 5th, he voided approximately 5 ozs. of dark red urine containing a heavy tarry black sediment. A few hours later he passed 1 oz. of pale yellow urine containing much less sediment. On the 6th and 7th, he passed a little more than an ounce of urine a day; after this the volume slowly increased until by October 12th he was passing over 8 pints of pale clear urine. During the first week in hospital, the patient suffered greatly from nausea and vomiting. He gradually became more drowsy, his speech became thick, his breadth uriniferous, and his face more and more oedematous. He complained of pain in the region of the right kidney, of considerable thirst, and general restlessness. During the second week the condition gradually improved, and by October 12th he was on the way to recovery. During the fourth week he was sufficiently well to be invalided to England. The jaundice cleared up rapidly after admission to hospital, there was little oedema except in the face and a moderate accumulation of abdominal fluid early during the second week. The blood pressure remained approximately 120/60 throughout. The temperature was 104·5° F. on admission, fell rapidly to 100° F., and after the second day remained normal. The patient was given fluids, salt, glucose, and sodium bicarbonate mostly by rectal tubes during the first two weeks. Frequent examinations of the

blood and urine were made throughout the course of the disease. The bio-chemical study is of interest in showing the lack of any acidosis, the very small amount of haemoglobin excreted by the kidneys, the extreme rapidity with which the haemoglobin disappeared from the general circulation, the extreme degree of nitrogen retention in the blood, and the striking impairment of renal function followed by recovery. Results of examination of the blood and urine are presented in tables.

[Records of this case are of considerable interest and importance and should be compared with those recorded by OWEN and MURGATROYD.]

W. Y.

OWEN (D. Uvedale) & MURGATROYD (Frederick). **Clinical and Chemical Observations on Two Cases of Blackwater Fever.**—*Ann. Trop. Med. & Parasit.* 1928. Dec. 28. Vol. 22. No. 4. pp. 503–530. With 6 figs. [48 refs.] [Liverpool School of Tropical Medicine, Liverpool.]

Clinical details are given regarding two cases of blackwater fever which occurred in the hospital of the Liverpool School of Tropical Medicine. Quantitative estimations of the haemoglobinaemia, bilirubinaemia, and the blood non-protein nitrogen were made. Especial attention was given to the urine, and information is supplied on the following points, viz., quantity of urine passed, specific gravity, colour, reaction, deposits, spectroscopic appearances, quinine excretion, bile pigments and salts; in addition, quantitative examinations of the urobilinogen, albumin, urea and haemoglobin were made. Every specimen of urine passed was examined in this manner, and the data are collected into two tables. Neither case proved fatal, although one had suppression of urine for thirty-four hours.

The following facts are worthy of notice :—

1. The haemoglobinaemia in each case had apparently returned to normal within forty-eight hours of the commencement of haemoglobinuria.

2. Both cases gave a direct and indirect positive van den Bergh reaction.

3. Definite but temporary renal inadequacy was demonstrated in both cases by the non-protein nitrogen determinations.

4. No justification was found for the view that the specific gravity of the urine in blackwater fever was abnormally low.

5. Bile pigments and bile salts were absent in the urine in both cases.

6. Detailed records of the urinary excretion of urea for twenty-four days are given. The authors were unable to find in the literature any previous quantitative records.

7. The output of urobilinogen in the urine gave no indication of the onset of haemoglobinuria, since it was found to be no higher before the onset than in many cases of uncomplicated malaria. Excessive output did not extend into convalescence.

8. In view of the serious objections to all previous methods used for estimating haemoglobinuria in blackwater fever, the authors devised a new method, based on the quantitative estimation of the iron content of the urine. This method is described in detail in the paper where emphasis is placed on its applicability to urines, however old they may be.

W. Y.

McCutcheon (Oliver). **Note on the Endemicity of Blackwater Fever in the Bengal Duars.**—*Report on a Mosquito-Malaria Survey of the Duars Tea Gardens.* pp. 67–75. With 2 charts. [13 refs.]

Blackwater fever has been known in the Bengal Duars for about forty years. Particulars of 382 cases occurring in the Duars during the five years 1921–25 have been placed at the author's disposal. These cases are classified, in a table, according to nationality, and the number of deaths which occurred in each group are given. Of 382 cases, 217 were Bengalis with 52 deaths. The author remarks that "this number is absolutely large and enormous in proportion to the 'babu' population in the Duars, which probably does not exceed 1,000." Sex appears to have no influence on the incidence of the disease. Information is given regarding the seasonal incidence of malaria and blackwater fever and also of the length of residence of the patients in the Duars before they developed blackwater.

W. Y.

Cort (E. C.). **Epidemiology of Blackwater Fever in Siam.**—*Amer. Jl. Trop. Med.* 1929. Mar. Vol. 9. No. 2. pp. 105–115. With 1 text fig. [26 refs.] [McCormick Hosp., Chiengmai, Siam.]

After a very brief review of (*a*) the part played by malaria in blackwater fever, (*b*) the production of a haemolysin, (*c*) the rôle of quinine in the production of haemoglobinuria, and (*d*) the question of a specific infectious agent, the author passes to a consideration of certain epidemiological observations made by him in Siam.

The following summary is given :—

"Blackwater fever, before unknown in Siam, appeared in Chiengmai in epidemic form.

"The first epidemic was confined to practically one city block and to three family connections.

"The second epidemic occurred in a boys' school 4 miles from the city.

"A short time after the first epidemic sporadic cases, 12 in all, began to appear.

"The evidence would seem to point to a specific factor, supposedly a malaria parasite, but a variety capable of elaborating a potent hemolysin.

"Prevention of relapse in a series of cases with neoarsphenamine without quinine would seem to suggest that this parasite was a variety of *Plasmodium vivax.* But it should be noted that these same observations from treatment alone might point to a spirochaete or a leptospira."

W. Y.

Hillel-Yofé. De la fièvre hémoglobinurique en Palestine. [**Blackwater Fever in Palestine.**]—*Rev. Méd. et Hyg. Trop.* 1929. July–Aug. Vol. 21. No. 4. pp. 105–114. [15 refs.]

In this address the author conveys some impressions resulting from a study of the 260 cases of the disease he has had to deal with during 37 years work in Palestine.

Of the 146 cases in which it was possible to go into the history of quinine in only 3 could he be certain that this drug had not been taken before the paroxysm. The author's observations on this point therefore support the general view that with rare exceptions the attack is precipitated by quinine. Of over 200 cases, only 4 were persons indigenous to the country. Information is given regarding the

seasonal, sex and age incidence of the disease. The mortality varied greatly in different years according to the general conditions, the season and the treatment. The paper closes with a discussion of the treatment of the disease.

W. Y.

LICHTENSTEIN (A.). Bijdrage tot de kennis van de pathogenese der zwartwaterkoorts. [**The Pathogenesis of Blackwater Fever.**]—*Geneesk. Tijdschr. v. Nederl.-Indië.* 1929. Feb. 20. Vol. 69. No. 2. pp. 138–148. [30 refs.]

Founding his opinion upon clinical reports of cases of blackwater fever in which the characteristics of haemolytic jaundice were found (lowered resistance of the erythrocytes against hypotonic salt solution, bilirubinaemia without bilirubinuria, marked urobilinuria, the absence of bile salts in the urine), the author comes to the conclusion that blackwater fever is an acute haemolytic jaundice. The aetiologic factor is always malaria infection ; either the malaria parasites may, under so far unknown circumstances, acquire haemolytic properties, or some other specific agent is necessary to cause haemoglobinuria in the malaria patient. In cases of moderate haemolysis the bile producing organs are able to alter all the affluent haemoglobin into bile pigment ; in case of abundant liberation of haemoglobin, however, the excess has to be excreted by the kidneys, causing the symptom of haemoglobinuria.

Blackwater fever may occur without urobilinuria ; in such cases however, large quantities of urobilin are found in the faeces.

W. J. Bais.

HEWETSON (W. M.). **Blackwater Fever. An Interpretation of the Symptoms with a Suggestion for Treatment.**—*Jl. Trop. Med. & Hyg.* 1929. June 15. Vol. 32. No. 12. pp. 157–165. With 7 charts. [3 refs.]

This paper is a condensation of an article read to the local division of the British Medical Association four years ago. The author quotes THOMSON as stating that " The haemoglobin is evidently highly toxic, as witnessed by the severe rigors which accompany its liberation into the blood. There seems little doubt that the toxicity of the haemoglobin and the products of other dead tissue cells probably aid in the production of the rigors."

The author challenges these statements. He believes the symptoms are principally due to the toxicity of the bile. He finds that the appearance of the icterus and grave symptoms is simultaneous and in corresponding degree. The symptoms of a typical case are described in detail, and clinical notes of several cases are recorded. Blackwater fever is contrasted with such diseases as yellow fever and anaplasmosis in cattle in which jaundice is a prominent symptom. The main principles of the treatment in blackwater, apart from the usual attentions to heart, etc., are : (1) evacuant, and (2) diluent.

(1.) Mercury in one form or another is used. The object of mercury, which acts chiefly on the duodenum and upper part of the jejunum, is to sweep the bile rapidly away from the upper intestine and thus prevent reabsorption.

(2.) The author believes that the value of intravenous or subcutaneous injections is due not so much to their action in flushing the kidneys, as to their flushing the liver.

The paper concludes with a theoretical discussion of the desirability of operation in selected cases. He believes that in cases other than suppression, particularly in fulminating ones in which the chance of life is excessively remote, a large intravenous saline should be given immediately. If no definite satisfactory result is seen in about four hours, the gall bladder should be opened. The author states that if this operation is performed at all, it must be done early and not after fatal toxic effects have told on the heart, liver and kidneys. He anticipates that the general effects of cholecystostomy would be an immediate cessation of the disastrous restlessness and vomiting and a clearing of the mental symptoms ; there would be none of those " so-dangerous " relapses.

W. Y.

PATERNI (Ludovico). Il rene emoglobinurico. (**Haemoglobinuric Kidney.**)—*Riv. di Malariologia.* 1928. Sept.–Oct. Vol. 7. No. 5. pp. 659–689. [24 refs.] [English summary pp. 844–845.] [S. Spirito Hosp., Sassia.]

The author has studied the kidneys of four cases of blackwater fever. He insists on the relative frequence of changes in the vessels and circulation of the glomeruli ; these changes are not of the nature of a real nephritis. The author does not consider that the mechanical blocking of the kidneys is responsible for the anuria. He considers that the presence of casts in the kidney is rather the result than the cause of the oliguria ; and that the disease itself exerts some influence on the function and structure of the kidneys, the pathological changes of which do not depend upon the elimination of haemoglobin.

W. Y.

HOEPPLI (R.). Ueber degenerative Nierenveränderungen bei Schwarzwasserfieber. [**The Degenerative Renal Changes in Blackwater Fever.**]—*Arch. f. Schiffs- u. Trop.-Hyg.* 1929. June. Vol. 33. No. 6. pp. 322–328. [10 refs.] [Inst. for Ship & Trop. Diseases, Hamburg.]

After referring to some of the previous work on this subject, the author describes in detail his own observations on the kidneys of nine persons who died of blackwater fever at Hamburg. The following summary is given :—

Among the nine cases of blackwater, the kidneys of two showed a slight degree, and those of seven a more or less pronounced degree, of degeneration of the epithelium of the convoluted tubules, with displacement of epithelial cells.

The displaced epithelium played a substantial part in the formation of the casts in the tubes of Henle and collecting tubes.

An escape of blood into the renal tubules was seen in only four cases and then only in small amount.

These investigations do not support the view that the kidneys constitute the site of haemolysis in blackwater fever.

[BARRATT and the reviewer recorded similar observations twenty years ago, but Hoeppli is apparently ignorant of the fact, as no reference to their papers is given.]

W. Y.

TALIANIDIS (Pr. Panos). Zur Behandlung des Schwarzwasserfiebers. Vergleichende Untersuchungen aus dem Zentralkrankenhaus für Flüchtlinge in Saloniki. [**The Treatment of Blackwater Fever. From the Refugees' Hospital, Salonica.**]—*Arch. f. Schiffs- u. Trop.-Hyg.* 1928. Dec. Vol. 32. No. 12. pp. 607–609.

In the Central Hospital for Refugees at Salonica there have been during the last four years 80 cases of blackwater fever. In every case spirochaetes were searched for unsuccessfully. Most of the attacks occurred in the autumn months (37 cases), in January 12 cases, and only very few in the remaining months. The majority (57) were manual workers and men in a poor state of health ; amongst these the mortality was high (49 per cent.) : 27 cases were women and the mortality was 18 per cent. Most of the patients had taken quinine, some in small quantities daily, the remainder only when they had fever. Occasionally, single large doses were taken either in the evening or during the morning.

From the point of view of prognosis, it seems of importance whether the blackwater was accompanied by fever and whether or not parasites were present in the peripheral blood. Of 8 febrile cases with parasites, 5 (62·5 per cent.) died ; whilst of 51 febrile cases without parasites, only 18 (19·8 per cent.) died. The final cause of death in all cases was uraemia. The results of attempts at treatment can be summarized as follows :—

1. Of the 10 cases which were treated symptomatically with serum, haemostatics, cardiac tonics, etc., 8 (80 per cent.) died.
2. Of the 30 cases which were treated with quinine, 21 (70 per cent.) died.
3. Of the 14 cases which were treated with plasmochin, 2 (14·3 per cent.) died.

W. Y.

ARAVANTINOS (Anastase). Pathogenése et traitement de l'hemoglobinurie paludéenne. [**Pathogeny and Treatment of Malarial Haemoglobinuria.**]—*Medicina Países Cálidos.* Madrid. 1929. July. Vol. 2. No. 4. pp. 334–335.

The author believes that there is only a malariál haemoglobinuria and that the term bilious haemoglobinuric fever should disappear, as it conveys the impression of a disease apart from malaria.

Haemoglobinuria is due to an abnormal activity of the reticulo-endothelial system. This hyper-activity is the result of more or less prolonged malaria and manifests itself clinically by haemoglobinuria, which is precipitated either by an attack of malaria or by the administration of quinine or some other drug. In the first case quinine cures the attack of haemoglobinuria, whilst in the latter it is contraindicated.

As a result of experience in the treatment of 15 cases, the author states that the treatment of blackwater should take two lines. First the treatment of the haemoglobinuric paroxysm, and secondly the

treatment of the malaria. For the haemoglobinuria he advises the intravenous injection of a colloid with a view to blocking the reticulo-endothelial system. Such a procedure sometimes acts like a charm. With a view to preventing the suppression of urine, he advises the intravenous injection of novasurol or the intramuscular injection of salyrgan. He states that the kidneys in blackwater tolerate both these mercurials well. With regard to the treatment of malaria, which is necessary in order to prevent a relapse of haemoglobinuria, the author recommends desensitization of the patient in the following manner: An intravenous injection of a small quantity, ·05 gm. to ·1 gm., of quinine should be given between two injections of a colloid, with a view to blocking the reticulo-endothelial system. He declares that usually the first injection provokes an attack of haemoglobinuria. Two or three days later a second injection of quinine is given. This should be larger than the first if the haemoglobinuria was not intense. In the ordinary course three or four injections suffice to desensitize the patient and then quinine can be given by the mouth in the ordinary way.

W. Y.

COOKE (W. Ernest) & WILLOUGHBY (Hugh). **Note on the Use of Intravenous Sodium Bicarbonate Solution in the Treatment of Blackwater Fever.**—*Lancet.* 1929. Feb. 16. pp. 334–335. [3 refs.]

Details are given of two cases of blackwater fever which were treated by intravenous injections of sodium bicarbonate solution, as recommended by HANSCHELL [see this *Bulletin*, Vol. 24, p. 667].

The following are the comments on the cases:—

"1. Both cases were negative to tests for paroxysmal haemoglobinuria.

"2. As both patients were of continental nationality and accustomed to alcohol, champagne was given daily in small quantities without adverse effects.

"3. Case 1 had two intravenous transfusions, and even though he was an older man and had had in addition the shaking and disturbance of an ambulance journey at the height of the attack, his urine cleared more rapidly than did that of Case 2, and he did not have such severe haemolysis. In his case the second transfusion, indicated on account of the subnormal temperature and lack of appreciable improvement in the urine, may have been the determining factor in obviating the persistence of the haemolytic process. In Case 2 the urine took longer to clear and there was a more severe haemolysis, but only one transfusion was given. The second transfusion was withheld, as the temperature was approaching normal and rectal salines were being well retained.

"4. Both cases gave a negative result to the Wassermann test, and there were no signs of any intercurrent disease.

"5. Judging from the results of these two cases it appears that immediate transfusion with sodium bicarbonate solution is indicated in all cases of blackwater fever to curtail the duration of the attack, to prevent blockage of the kidney tubules, and to avoid suppression of urine.

"6. More than one transfusion may be needed and should be given when there is no appreciable improvement within the first 24 hours. The need for further transfusions should be judged by the progress of the case.

"7. Although rigors and further haemolyses may occur after transfusion the effect on the urinary system fully justifies the measure, but should signs of anoxaemia appear blood transfusion must be employed instead, and this must be from a donor who is not taking quinine."

W. Y.

i. CHESTERMAN (Clement C.). **The Treatment of Blackwater Fever by Oral Sodium Bicarbonate.**—*Lancet.* 1929. June 29. pp. 1355–1356. [1 ref.]

ii. DONNISON (C. P.). **Bicarbonate for Blackwater Fever.** [Correspondence.]—*Ibid.* July 6. p. 47.

i. Details are given of a case of blackwater fever treated by large oral doses of sodium bicarbonate. The author states that the following points are of interest in this case :—

" (1) Both in the initial attack and the relapse the disappearance of haemoglobin from the urine, which was formerly acid, followed a few hours after it showed an alkaline reaction. (2) This was effected solely by the oral administration of sodium bicarbonate (and the citrates of lemonade) in doses up to 21 g. in 24 hours. Bilious vomiting which caused rejection of the first few doses was controlled by frequent sips of this alkaline fluid. (3) The relapse on the tenth day followed a couple of 0·6 g. (10 grains) doses of quinine when the urine was acid, whereas 1·2 g. (20 grains) was taken with impunity ten days later when the urine was kept alkaline. (4) Sodium bicarbonate in large doses is easily detected in the urine by the effervescence of $CO_2$ caused by the addition of an acid. The haemoglobin in such urine gives a very slight cloudy precipitate on boiling, whether previously acidified with acetic acid or not, possibly on account of the formation of alkaline haematin. Nitric acid, however, throws down a heavy cloud.

" Although in this case the illness was precipitated by quinine, it was definitely not a case of quinine haemoglobinuria, for the patient showed no sign of quinine idiosyncrasy before or after the attack. Moreover, its association with an enlarged spleen, bilious vomiting, and jaundice leaves no doubt that it was a case of true blackwater fever. The case can be written down as but of slight severity, only on the ground that it continued for so short a period, and it is suggested that the early treatment, within six hours of onset, definitely aborted what might have been an attack as serious as the first one.

" Such simple treatment is available immediately for any sufferer, however far removed from medical attention. The pre-blackwater state should similarly be treated with small doses of quinine combined with generous doses of alkali taken some hour or so before. It is hoped that medical men practising in endemic areas will make known the possibility of such treatment of this dangerous and much dreaded disease, and will report in confirmation or failure as results occur, for any single practitioner has as a rule to wait some years in order to collect a series of cases."

ii. This letter is written in support of the statement by CHESTERMAN regarding the value of oral administration of bicarbonate of soda in blackwater fever. The author apparently treated two cases successfully by this means.

W. Y.

BELLGARD (S. J.). **Observations on 8 Cases of Blackwater Fever, treated with Serums and Alkalies.**—*Indian Med. Gaz.* 1928. Oct. Vol. 63. No. 10. pp. 573–574.

The author gives a few statistics showing that blackwater fever cases were most common when malaria was at its height. About 90 per cent. of the malaria infections seen by him were *Plasmodium falciparum.* Details are given of a method of treatment consisting of intravenous

injection of anti-streptococcic serum and oral administration of large doses of alkali, which, in the author's opinion, was of benefit to the 8 patients on whom it was tried.

W. Y.

Low (G. Carmichael), Cooke (W. E.) & Martin (P. H.). **Blood Transfusion in Blackwater Fever.**—*Lancet.* 1928. Sept. 29. pp. 645–647. [5 refs.] [Hosp. Trop. Diseases, Endsleigh Gardens, London.]

Low states that an experience of 28 years, both abroad and at home, has lead to the conclusion that, apart from careful nursing and attention, there is no specific treatment for blackwater fever. Many drugs have been lauded from time to time, only to fall into disfavour and disuse.

The main causes of death in blackwater fever are: (1) syncope; (2) acute haemolysis; (3) suppression of urine; (4) hyperpyrexia; and (5) asthenia. In the last type of case, the urine clears, but the patient does not improve, the blood does not regenerate, and gradually a semi-comatose condition intervenes and death takes place.

In a recent case of this type blood transfusion was tried. A detailed account of the case is given. The patient, who was apparently at the point of death, made a good recovery. Reference is made by Manson-Bahr and Sayers [this *Bulletin*, Vol. 24, p. 650] to the record of a somewhat similar case. It is concluded that transfusion of whole blood at the time of, or immediately after, an attack of blackwater fever provides us with a new and useful therapeutic measure in the treatment of certain types of the disease.

With regard to the actual and theoretical effects of blood transfusion in blackwater fever, the authors write as follows:—

"1. It undoubtedly treats anoxaemia symptomatically by helping to replenish the patient's supply of red blood cells.

"2. It acts as a stimulus to blood cell formation. There may or may not be an adequate stimulus already provided, but in our case the rise in the number of red cells per cubic millimetre coincided with the time at which blood rejuvenation occurs in other diseases after blood transfusion.

"3. The urine in blackwater fever is loaded with albumin. The introduction of healthy blood plasma is probably advantageous.

"4. It dilutes the haemoglobin and the bile pigment already free in the plasma, and so may aid the kidney in its work of excretion.

"5. It replenishes blood-volume without unduly increasing the amount of sodium chloride. Baker & Dodds[4] have shown in their work on obstruction of renal tubules during the excretion of haemoglobin that a percentage of more than about 1 of sodium chloride is a very important factor in causing coagulation of haemoglobin, and therefore tends to cause suppression of urine. Further, Dr. J. G. Thomson, in conversation with one of us (P.H.M.), has recounted how disappointing and sometimes even disastrous were his results in treating blackwater fever in S. Rhodesia with intravenous salines. This was, of course, before the publication of the work of Baker & Dodds in 1925[4].

"6. It provides the most natural antidote possible to the acidosis and the acid urine which Baker & Dodds have shown in the same paper to be so very important in causing coagulation of the liberated haemoglobin.

"It is noteworthy that in our case, in spite of the alkali treatment which was carried out from the start of hospital treatment, the urine was still acid to litmus on the second day—after nearly 24 hours' treatment.

[4] S. L. Baker and E. C. Dodds: *Brit. Jour. Exper. Path.*, 1925, vi, 247. See this *Bulletin*, Vol. 24, p. 664.

"7. It provides such antibodies as the donor may have in his blood against organisms which are likely to cause intercurrent infection—e.g., lung complications.

"8. It does not appear to lead to further haemolysis, provided the bloods are carefully matched. Autoagglutination has been observed by us in severe malarial infections, and demands the utmost care to keep the temperature of the ingoing blood and of the receiving limb as near normal as possible.

"9. Whether there is any specific therapeutic action against *P. falciparum* is very doubtful."

W. Y.

STERCKX (F.). Un cas d'hémoglobinurie grave traité par transfusion du sang. [**Case of Severe Haemoglobinuria treated by Blood Transfusion.**]—*Ann. Soc. Belge de Méd. Trop.* 1928. Sept. Vol. 8. No. 2. pp. 161–163.

The patient was injected on July 31st, 1927, with 250 cc. of serum glucose shortly after he had commenced an attack of blackwater. Examination of the blood revealed an intense malarial infection; the author remarks that three-quarters of the red cells contained parasites. The next day, as the urine was still black, the injection of serum was repeated. At midday the contents of one ampoule of antivenom serum was injected intravenously. The state of the patient was now worse; the temperature remained at 39·5 °C., the urine was black and scanty and the pulse feeble. The injections of physiological serum were continued, and injections of caffeine and camphorated oil given. During the following night of August 2nd, the urine cleared temporarily and the previous treatment was continued, the injections of serum being repeated at intervals of two to three hours. The next day the urine was very scanty with an enormous deposit. An intramuscular injection of antivenom serum was given. About 1 p.m. bilious vomiting set in, and the pulse was hardly perceptible. The patient was considered to be on the point of death. In view of the desperate situation, the author decided to try transfusion of blood: 100 cc. of blood was mixed with a solution of 10 per cent. citrate and an attempt made to inject it intravenously, but after 45 cc. had been injected, the attempt had to be abandoned owing to obstruction of the needle. Violent reaction followed, and it was feared that the patient was dying. Subsequently, however, the patient passed fair quantities of clear urine. It was decided to give a second transfusion shortly after midnight, and 100 cc. of blood was given. After this the patient began to recover. The author considers that the transfusion saved him.

W. Y.

CAMBESSEDÈS (H.) & WESTPHAL (F.). Fièvre bilieuse hémoglobinurique et quinine. [**Blackwater Fever and Quinine.**]—*Rev. Méd. et Hyg. Trop.* 1928. July–Aug. Vol. 20. No. 4. pp. 97–102. With 1 chart.

Details are given of a case of blackwater fever which was treated with quinine. During his first tour in the Ivory Coast in 1923–1924, the patient, who was 29 years of age, had two slight attacks of malaria. During his second tour in 1926 he had rather severe attacks of malaria, followed in June of the same year by a mild attack of blackwater fever. During this tour he had taken prophylactic quinine regularly. On March 17th, 1927, he set sail for France, and when on the ship he stopped quinine. On April 4th, 1927, he commenced with a series of acute attacks of malaria and recommenced quinine 1 gm. per day for 3 days. *P. vivax* was found in the blood. Up to April 10th he took 0·5 gm. quinine daily and then 0·25 gm.

to April 22nd. On the morning of this day, as he didn't feel so well, he took 0·5 gm., and about midday another dose of 0·25 gm. At 1 p.m. the temperature rose rapidly to 40° C. and at 3 p.m. he passed black urine. During the next two days he exhibited the classical signs of blackwater fever. On April 23rd the patient had two paroxysms of fever, one at 6.45 a.m. and the other at 2 p.m. At 5.30 p.m. an injection of 0·25 gm. of quinoform was given: half an hour later there was a further paroxysm, and at midnight a second injection of 0·2 gm. of quinoform was given. At 2.30 a.m. on April 24th there was another very violent paroxysm, and a third injection of quinoform was given at 7 a.m. An hour later there was a further paroxysm, but it was distinctly milder. Further injections were given, but were not followed by paroxysms and the temperature gradually subsided to normal.

There follows a brief discussion of the case, which adds nothing definite to knowledge. [Somewhat similar cases were recorded twenty years ago by BARRATT and the reviewer, and others are to be found scattered throughout the literature.]

W. Y.

GENOESE (Giovanni). Contributo allo studio della emoglobinuria nella malaria dei bambini. [**Blackwater in Children.**]—*Pediatria.* 1928. May 1. Vol. 36. No. 9. pp. 465–480. [Inst. Clin. Pediatrics, Univ., Rome.]

The case here recorded is interesting since blackwater fever is believed to be rare in children. The patient, a boy 5 years of age, had never been far from Rome, and first came under observation for pleurisy with effusion. The Von Pirquet test was positive. A month later a sharp attack of malaria lasting 24 hours was succeeded after an apyrexial interval of 8 hours by a second chill and dark red urine was passed. Jaundice was present and the child was obviously very ill. Subtertian parasites were found in the blood. He was given large doses of calcium chloride (1 gm.) and injections of camphor and digalen, and quinine hypodermically. The urine became almost black and the quinine injections were replaced by the tannate (1 gm.) *per os*. The second attack of fever lasted for 26 hours, and after an apyrexial interval of 10 hours was succeeded by a third. Improvement then set in and convalescence was established in a fortnight, the quinine and calcium being given throughout. The author lays great stress in the value of calcium chloride in large doses in the treatment of malarial haemoglobinuria, in which condition, he states, the blood calcium is below normal. He has given as much as 8–10 gm. in the day.

H. Harold Scott.

GREY (C. G.). **Quinine Intolerance in an African Child.**—*West African Med. Jl.* Lagos. 1928. July. Vol. 2. No. 1. p. 96. With 1 chart facing p. 109.

The patient was the daughter of a native hospital dispenser at Opobo. Up to the age of four, she was given quinine in one grain doses, without ill-effects, when she had "fever."

In 1927, when she was four years of age, she was given 2 grains of quinine in a single dose on one occasion when she was out-of-sorts. Four hours later she had a rigor, the temperature rose to 105° F., and haemoglobinuria occurred; the condition lasted two days. In February, 1928, she was again given quinine because of fever and again she developed haemoglobinuria; this attack lasted four days. In May the author gave her two tablets of plasmochin compound (plasmochin and quinine) not because she had fever, but simply with a view to testing the drug. Within four hours she had a rigor and passed blood-red urine: the temperature rose to 101·4° F., and the attack lasted 24 hours.

W. Y.

DUPUY (H.). Un cas de fièvre bilieuse hemoglobinurique de nature nettement paludéenne, traité par le stovarsol. [**A Case of Blackwater Fever treated by Stovarsol.**]—*Bull. Soc. Path. Exot.* 1928. Oct. 10. Vol. 21. No. 8. pp. 636–637.

Details are given of a case of chronic malaria, in which the patient suddenly developed blackwater after he had undergone 4 days quinine treatment. Quinine was stopped and the patient treated with intravenous injections of 30 cc. of antivenom serum. First the urine became more clear, but a day or so later it became black again. Examination of the blood showed numerous schizonts of *Plasmodium vivax*. The injections of serum were continued, but the condition of the patient grew worse and the anaemia rapidly increased. On the 5th day, the urine had become clear, but the temperature remained high. A dose of 1 gm. of stovarsol was given daily for 3 days, and then ½ gm. daily for 9 days. The temperature rapidly returned to normal, and the patient quickly recovered.

W. Y.

NAG (S. C.). **Retention of Urine in Blackwater Fever.**—*Indian Med. Gaz.* 1929. Sept. Vol 64. No. 9. pp. 505–506.

The author states that he has encountered retention of urine apart from suppression in 5 of 36 cases of blackwater fever. He concludes that, while suppression of urine is likely to occur in a case of blackwater fever when sufficient fluid is not given, retention of fluid is not uncommon when, during the course of the disease, a large amount of fluid is given and the patient is exhausted.

W. Y.

WHITMORE (Eugene R.). **The Blood in Blackwater Fever.**—*Seventeenth Ann. Rep. Med. Dept. United Fruit Company, Boston, Mass.* 1928. pp. 123–137. [7 refs.]

This paper deals with Laboratory Studies on cases of blackwater fever, and more particularly with blood examinations. The study consists of typing, coagulation time determinations, studies of the blood picture, tests of the resistance of the red blood cells to hypotonic salt solution, saponin, and cobra venom, tests for hemolysins and quinine haemolysis, and blood chemistry determinations. All the patients were Cuban, except one Cuban mulatto and one Barbadian.

*Typing.*—Scanty reports of blood typing in blackwater fever indicate that there is no relation between blood type and susceptibility to the condition.

*Flocculation test.*—The results of the Kahn test are set down in tables; positive results occurred both in controls and in the blackwater cases, and probably these are syphilitic.

*Coagulation time.*—This was determined by the capillary tube method. In no case was there any departure from the normal. The results of the examination of the individual cases are given in tables.

*Resistance of the erythrocytes to hypotonic salt solution.*—The ordinary technique was followed, and it was found that when fresh red cells were washed and used at once, or when fresh blood was dropped directly into the tubes, there was no difference in the resistance of the red cells of the cases and those of the controls.

*Resistance of the erythrocytes to saponin.*—For these determinations, dilutions of saponin from 1 : 35,000 to 1 : 46,000 were used, and measured quantities of fresh washed red cells were added to each tube. No difference was found between the resistance of the blackwater red cells and those of the controls.

*Resistance of the erythrocytes to cobra venom.*—For these determinations, dilutions of cobra venom of 1 : 10,000, 1 : 15,000, 1 : 20,000 1 : 30,000, and 1 : 40,000 were used, and a measured quantity of fresh washed red cells added to each tube ; here again, no difference was found in the resistance of the blackwater red cells and those of normal persons.

*Test for serum haemolysins and quinine haemolysis.*—The cold haemolysin test was negative, as also was Ehrlich's test. The question of the part played by quinine in blackwater fever renders it important to consider whether quinine exerts any haemolytic action on the red cells of cases and subjects of blackwater fever. In no case did the author find any indication that quinine hydrochloride in a dilution of 1 : 2,000 exhibited a haemolytic action either in mixtures of serum and red cells or in mixtures of red cells and salt solution. In two cases, 0·2 cc. of 1 : 1,000 solution of quinine hydrochloride was injected intradermally ; there was no reaction of any kind.

*Blood picture.*—The study of the blood picture of a severe case is shown in tabular form. In this table information is given regarding the red cell count, the leucocyte count and differential leucocyte count.

*Blood chemistry.*—As the author was not equipped for blood chemistry determinations in 1927, he attempted to preserve whole blood by the addition of 20 mgm. of sodium fluoride per cc. ; the sera were placed in ampoules and carried back to Washington for study. The results of the examinations of these bloods and sera are recorded in a table from which it appears that it is not possible to get satisfactory results from determinations on such material when kept for a period of several months. In 1928, the non-protein nitrogen, uric acid, urea, creatinine, and sugar determinations were made as soon as the blood was taken ; the alcohol-ether extracts for cholesterol and lecithin were made promptly, evaporated to dryness, and the dried residues taken to Washington and examined there. The trichloracetic acid filtrates of the sera were carried to Washington, where the calcium and inorganic phosphorus determinations were made ; and whole blood was also taken to Washington and the phosphorus determinations made there. The results of the 1928 determinations are also given in a table.

*Carbon dioxide combining power of the blood plasma.*—It has been noted by Berghausen (1912) that the blood serum contained haemoglobin when blood was obtained for a Wassermann by constriction above the elbow—producing passive congestion. Berghausen studied the haemolytic action of carbon dioxide and also of the salts in the blood ; from this work he suggested that cold, trauma, and passive congestion, which lead to an attack of haemoglobinuria, may be associated with the production of an excessive acidity in the tissues, and that the organic acids thus formed may play a part in the haemolytic process.

Applying this idea to blackwater fever, Whitmore considered it advisable to examine the blood of cases in order to determine whether there was an association of "acidosis" in this condition. He investigated the carbon dioxide combining power of the blood plasma in two cases of blackwater and obtained evidence of mild acidosis in both cases. The article concludes as follows :—

"No conclusions are drawn, and no suggestions are made. It is recognized that more material must be collected before one is justified in making any suggestions. The work is in progress ; and this is merely a progress report."

W. Y.

FIESSINGER (Noel) & WALTER (Henry). Les procédés modernes de bilirubimétrie sanguine. [**Modern Procedures of Bilirubin Estimation in Blood.**]—*Ann. de Méd.* 1928. July. Vol. 24. No. 2. pp. 178–201. [53 refs.]

Since the classical work of GILBERT, HERSCHER and POSTERNACK (1903) bilirubimetry has become a current method for determining the titre of bilirubin retention in the blood. Three factors can intervene to modify cholaemia. There may be changes in the amount of pigmentogenous material available, e.g., diminution as in certain anaemias, or augmentation as in the case of haemolytic icterus ; there may be damage of the biliary function of the hepatic cells ; or there may be obstruction in the biliary passages with reflux of bile into the blood. In all cases of cholaemia, bilirubimetry is of great importance, not only because it allows of the expression by a definite figure of the degree of retention, but also because it discloses the existence of cholaemias clinically indistinguishable. The various methods which have been devised for the purpose, particularly those of van den Bergh and Meulengracht, are described in detail. This is followed by a critical consideration of the methods and a discussion of the indication for their use. [The paper is of a somewhat technical nature, and should be consulted in the original by those specially interested in this subject.]

W. Y.

NOCHT (B.) & KIKUTH (W.). Ueber hämolytische Chininwirkungen. [**On the Haemolytic Action of Quinine.**]—*Arch. f. Schiffs- u. Trop.-Hyg.* 1929. July. Vol. 33. No. 7. pp. 355–375. [12 refs.] [Inst. for Ship & Trop. Diseases, Hamburg.]

The following summary is given :—

1. The haemolytic action of a specific amboceptor is not increased by quinine *in vitro*. On the contrary, quite small doses of quinine expedite markedly the action of haemolytic amboceptor in the animal organism.

2. The strengthening influence of quinine on specific amboceptor haemolysis in the organism was observed regularly in dogs, cats, and rabbits. A similar action was not found in the case of other drugs such as cinchona, antipyrin, and plasmoquin.

3. As in the case of the haemolytic amboceptor so was the haemolytic action of cobra venom and lysozithin definitely increased in the organism by quinine.

4. The haemolytic action of amboceptor, of cobra venom, and of lysozithin on washed red cells *in vitro* was inhibited by the addition of cholesterin.

5. In the sera of a case of blackwater fever and of two syphilitics infected with malaria there was an especially low content of cholesterin. These sera had the capacity of limiting the haemolytic components of cobra venom *in vitro* to a less extent than the sera of healthy persons and of cases of malaria, whilst the haemolytic action of lysozithin and of amboceptors were somewhat inhibited by all sera in equal degree.

6. In rabbits, which had been fed with cholesterin for a long time, the haemolytic effect resulting from intravenous injection of amboceptor or lysozithin was definitely less than that occurring in normal animals. This protective action of the cholesterin content could be eliminated or weakened by small doses of quinine, if the amboceptor or the lysozithin was injected simultaneously with quinine.

7. Intravenous injections of a colloidal solution of cholesterin had no influence on the haemolytic effect of amboceptor if it were administered shortly before, during or after, the amboceptor.

W. Y.

GOSIO (Renato). Ricerche sulla emolisina della emoglobinuria da chinino nei malarici. [**Researches on the Haemolysin of Quinine-Haemoglobinuria in Malaria.**]—*Policlinico.* Sez. Med. 1928. Oct. 1. Vol. 35. No. 10. pp. 538–555. [3 refs.] [Inst. of Clin. Med., Univ., Rome.]

This article deals with the question of haemolysis largely on the lines of GHIRON's experiments already recorded in this *Bulletin.* The report is in a very condensed form and refers to a large number of experiments, presented in tables, making abstraction difficult. The general principle involved was to place graded amounts of 1 per cent. quinine hydrochloride with serum and red corpuscles from normal or malarial subjects, in the thermostat at 38° C., for periods varying from 10 minutes to 3 hours and noting the time and concentrations in which haemolysis occurred. Thus: Varied amounts of the quinine solution, 2 drops of a 5 per cent. suspension of corpuscles from a case of subtertian malaria, were placed for 10 minutes at 38° C. with 2 drops of fresh haemoglobinuric serum; observations were made every 10 minutes, and it was found that after the first 10 minutes haemolysis was complete in the 0·9 concentration, after 20 minutes in 0·8, after 30 in 0·7, after one and after 3 hours in 0·6. Two other sera gave rather less haemolysis in the same times.

With normal sera haemolysis was not complete even in the 1·0 concentration after three hours. The experiments were repeated using, in place of fresh serum, that which had been dried for 24, 48, and 72 hours, and it was found that the haemolytic effect was little, if at all, changed, but heating to 54° C. rendered it inactive. Next, the globulins, lipoids, and other constituents of the serum were separated and it was shown that the haemolysis depended upon the globulin fraction, the others having no haemolytic action.

The whole is of great interest now that the subject of blackwater fever is receiving so much attention; for fuller details the original must be consulted.

H. Harold Scott.

ARNELL (O. R.). **Blackwater Fever and Quinine. A Statistical Note on the Hypothesis that Quinine is likely to cause or precipitate an Attack of Blackwater Fever.**—*Kenya & East African Med. Jl.* 1929. June. Vol. 6. No. 3. pp. 75–79.

It is difficult to summarize an article of this sort, and it should be consulted in the original by those interested. The Government Statistician comments as follows:—

"The recording of the results in the way suggested would enable them to be examined statistically by methods recently developed for dealing with the association of variates classed solely in alternate categories. The probabilities of independence can then be calculated.

"I think the suggestion to record the results in this way an excellent one."

W. Y.

BENEDETTO (de Luca). La plasmochina nell'emoglobinuria da chinino. [**Plasmochin in Quinine Haemoglobinuria.**]—*Riforma Med.* 1929. Aug. 17. Vol. 45. No. 33. pp. 1124–1126. [8 refs.]

Brief notes of fifteen cases are given to show that plasmochin is not only safe to prescribe, but certain in its action in patients who are suffering from blackwater fever or who have a return of malaria subsequent to such an attack.

H. Harold Scott.

RIGHT-SMITH (R. J.). **Recovery in Blackwater Fever after Blood Transfusion.**—*Med. Jl. Australia.* 1928. July 28. 15th Year. Vol. 2. No. 4. p. 120. [1 ref.] [Walter & Eliza Hall Inst. of Research, Melbourne Hosp., Melbourne.]

Clinical details are given of a case of blackwater fever which recovered after transfusion with 600 cc. of citrated blood.

W. Y.

RANJEVA. Traitement de la fièvre bilieuse hémoglobinurique par la collobiase de quinine. [**Treatment of Blackwater Fever by Collobiase of Quinine.**]—*Marseille-Méd.* 1928. Dec. 5. Vol. 65. No. 34. pp. 696–698. With 1 chart in text.

An account is given of a case of blackwater fever from which the author draws the conclusion that collobiase of quinine has a specific action on the disease.

W. Y.

MORALIS (Plutarque A.). Fièvre bilieuse hemoglobinurique provoquée par la quinine, chez un paludéen. [**Blackwater Fever provoked by Quinine.**]—*Monde Méd.* 1927. Aug. 15. Year 37. No. 713. pp. 760–763.

This article describes in considerable detail the clinical history of a case of blackwater fever, and calls for no special note.

W. Y.

## LEPROSY.

DE LANGEN (C. D.) & HERMANS (E.). La diffusion géographique et le problème de la lèpre. [**Geographical Distribution of Leprosy.**]—*Meded. Dienst d. Volksgezondheid in Nederl.-Indië.* 1929. Vol. 18. No. 1. pp. 208–220.

This instructive report was submitted to the Health Section of the League of Nations. The epidemiology of the disease is first discussed, from which the authors conclude that the disease is contagious through the entry of the bacillus through the skin, but only in certain phases of short duration ; they think superinfection may occur. Treatment and the regulation of the lives of lepers have become more important than isolation, as they allow the patients to be found in the early and favourable stages; a certain number will be cured and the serious cases will become less numerous. Special hospitals are required for the treatment of lepers in or near a large town ; these will also assist early diagnosis, and they consider that the chances of arresting the progress of the disease and even of cure is now great, and that 50 per cent. of fairly early cases can be cured, a few of them spontaneously. Lepra bacilli may be found after death in the glands of those who have lived with lepers without development of evident clinical lesions.

The different systems of dealing with leprosy, from compulsory or partial segregation with parole to no measures, are next considered, and they conclude that isolation of infective cases is of value, but the infectivity varies greatly and is decreased by treatment ; early diagnosis is of primary importance, while lepers should not be treated as criminals. They therefore recommend that in the Dutch East Indies there should be compulsory notification by medical men and the native chiefs, all suspected cases should be reported on by a committee with a lay president and two medical assessors in each area, and a positive diagnosis should be made when necessary on clinical grounds in the absence of bacteriological confirmation ; doubtful cases should be re-examined periodically, but those declared to be lepers should be sent to a hospital. A central leprosy institute with hospital, laboratory and library should be established at Batavia for research and training leprosy workers for other centres, and hospitals in central places, where intensive treatment will be carried out, and where voluntary cases will also be admitted and all infectious cases will be detained. Cases with a minimum degree of infectivity may live at home under supervision, as in the successful Norway system, and be reported on yearly. The highly susceptible children should not live with infective cases, and all children born in a leper institution should be separated from their parents as soon as possible, while certain professions should be forbidden to lepers. A knowledge of the real degree of infectivity of lepers should be spread among the public, and legal powers should be provided to allow highly contagious lepers to be isolated, while people coming from areas with high leprosy prevalence should be examined and cases of the disease prohibited from entry. There should be international collaboration to carry out such a system, with modifications to suit special conditions, and an international bureau if leprosy is to be properly dealt with.

L. Rogers.

DE MELLO (Froilano). Une croisade internationale, combattant la lèpre simultanément dans tous les pays, pourrait éteindre ce fléau en quelques décades. [**International Crusade against Leprosy with a View to its Extinction.**]—Nova Goa. 1928. Oct. 11 pp.

In this paper the frequency of slight easily-overlooked cases of leprosy and latent infection of the lymphatic glands is emphasized, and somewhat similar methods of dealing with the disease as in the foregoing abstract are advocated for Portuguese India, including treatment of early uninfective cases at home or as out-patients and isolation under comfortable conditions of infective cases. The author thinks the disease could be greatly reduced in five years and become an historical curiosity in three or four decades.

L. R.

WALKER (Ernest Linwood) & SWEENEY (Marion A.). **The Identity of Human Leprosy and Rat Leprosy.**—*Jl. Preventive Med.* 1929. July. Vol. 3. No. 4. pp. 325–333. [22 refs.] [George Williams Hooper Foundation for Med. Research, Univ. of California, San Francisco.]

The authors found acid-fast bacilli in 41 per cent. of 75 rats examined microscopically in San Francisco and also obtained cultures on Musgrave and Clegg's medium from the spleen, testis, glands, lungs or subcutaneous tissues of 24 out of 37 rats showing no gross lesions of rat leprosy. The organisms were pleomorphic like that found by them in leprosy cases previously described. They hold that the two micro-organisms are identical and that human and rat leprosy are the same disease and both are due to a soil actinomyces. They refer to the observations in the Philippines of PINEDA on finding acid-fast bacilli in the glands of apparently cured lepers, and consider them to be latent infections analogous to those of *Entamoeba histolytica*, and they think that this explains why a general soil infection with the lepra actinomyces produces so few cases of clinical leprosy.

L. R.

ARAUJO (H. C. de Souza). [In Portuguese & English.] Estudos sobre a lepra. III. Transmissão da lepra humana ao comondongo branco. **Studies upon Leprosy. III. Transmission of Human Leprosy to White Mouse.**—*Inst. Oswaldo Cruz, Supplemento das Memorias.* 1929. June 22. No. 8. In Portuguese pp. 113–117. With 6 plates. In English pp. 118–122. With 6 plates.

Of 30 white mice inoculated with emulsions of human leprosy nodules in 28 positive results were obtained, including nodules in 17, tumours in 5 and minute abscesses in 4. Animals inoculated with the same material tended to show similar lesions, but passages from mouse to mouse usually failed. Cholesterol was found in the tumours, but its origin is uncertain.

L. R.

LEPROSY NOTES. 1929. July. No. 6. 36 pp. Issued quarterly by the British Empire Leprosy Relief Association, 24 Cavendish Square, London, W. 1.

Information is recorded in this issue on leprosy in Nyasaland, where over 300 lepers are living in colonies in addition to others attending

clinics, and 30 have been discharged as free from symptoms. Alepol is used as causing less pain than moogrol. Leprosy relief in Cyprus is dealt with by Dr. Robert G. COCHRANE, the new secretary to the British Empire Leprosy Relief Association, and the introduction of modern methods in place of relying solely on compulsory isolation is advised for the 200 lepers believed to be present. Dr. E. A. NEFF deals with leprosy relief in Fiji with 409 cases, including those from other parts of Oceana. Alepol and sodium gynocardate stand pre-eminent in treatment and 55 patients have been passed by the medical board for discharge during 1928, or 14·7 per cent. of the treatment group and 12·4 per cent. of the mean population of the hospital. "Altogether the prospect of gradually but surely ridding these islands of leprosy seems bright, and one can honestly afford to feel optimistic." At the Lady Willingdon Leper Settlement, Chingleput, S. India, Dr. R. S. DONALDSON reports his preference for the ethyl esters over the whole oil; reactions and pains are much more frequent after the use of potassium iodide and potassium antimony tartrate intravenously, and adrenalin and ephedrine sulphate are needed in their treatment.

L. R.

CROZIER (G. G.). **Leprosy in Manipur State.**—*Indian Med. Gaz.* 1929. June. Vol. 64. No. 6. pp. 315–316.

Since work was begun in 1918, 173 lepers have been admitted to the Mission Leper Colony, thanks to the Manipur State officials becoming convinced that the disease was being cured and co-operating in seeking out and sending cases to the colony, and 40 mostly early cases have been discharged as apparently cured with very few returns. It is believed that in a few more years the State will be nearly cleared of the disease.

L. R.

TISSEUIL (J.). La lèpre aux îles de la Loyauté. Lifou. [**Leprosy in the Loyalty Islands.**]—*Bull. Soc. Path. Exot.* 1929. May 8. Vol. 22. No. 5. pp. 362–367.

The author deals with the incidence of leprosy in the Loyalty Islands, to which he referred briefly in his former paper on New Caledonia (*ante*, p. 611). A medical inspection in 1928 showed among 5,944 persons examined, 182 definite and 227 suspected lepers, 9 of the former and 61 of the latter being new cases, or a total of 6·87 per cent., including 3·81 per cent. of suspects; a very high rate. In 1912 only 3·44 per cent. had been found, and in 1919 5·70, indicating a steady increase in recent years, but the recent increase was only among suspected cases, so it is doubtful if a real increase has taken place. Isolation has not sufficed to reduce the disease, for new cases appear, so dispensaries should be provided for those whom it is useless or impossible to segregate.

L. R.

REISS (F.). **Leprosy.**—*China Med. Jl.* 1929. May. Vol. 43. No. 5. pp. 479–484.

This is a lecture at the Chinese Mission for Lepers in which the writer states that he considers individual treatment much more important

than isolation and that in China attempts at compulsory isolation causes cases to be hidden, while many escape, 40 per cent. in one place with an iron wall. Physicians well trained in dermatology are required to deal with the disease.

L. R.

HUIZENGA (Lee S.). **Legislation and Leprosy.**—*China Med. Jl.* 1929. May. Vol. 43. No. 5. pp. 486–490.

China has no national leprosy legislation and in some countries leprosaria are veritable prison houses, although it is now recognized that the disease is not nearly so communicable as tuberculosis and venereal disaese, Isolation laws therefore should be reconsidered, and with education to calm society other more humane legislation will become possible.

L. R.

SCHWARZMANN (B.). Beobachtungen ueber die Verbreitung der Lepra am Kaspischen Meer. [**Observations on the Spread of Leprosy on the Caspian.**]—*Zent. f. Bakt.* I. Abt. Orig. 1929. June 28. Vol. 112. No. 6/8. pp. 458–460. [16 refs.]

The author concludes from his study that leprosy is brought into the Caspian and Caucasus areas exclusively from Persia and the spread is assisted by the proximity of the sea. He thinks that fish eating and unhygienic conditions of living play a part in the incidence of the disease, and also that flies and mosquitoes may easily become infected from the excreta of lepers.

L. R.

PUENTE (José J.). Extracto de la memoria correspondiente al año 1928. Lepra, sífilis y venéreas. [**Leprosy, Syphilis and Venereal Diseases. From the Report of the Health Department, 1928.**]—*Semana Méd.* 1929. Aug. 8. Vol. 36. No. 32 (1856). pp. 402–406.

The leper census of the littoral Provinces of the Argentine has been continued and 1,687 cases have been noted. 25 per cent. of these were in Capital Federal and 20 per cent. (333) in Santa Fé. The figures are not regarded as accurate, for some cases in early stages have probably been overlooked, while a considerable number had been wrongly diagnosed as leprosy, such as vitiligo, keloid, psoriasis, traumatic neuritis, for example. The only hospital of any size where patients are received and treated is the Hospital Muñiz ; " the small leprosarium of Corrientes and the asylum of Córdoba cannot be considered of practical use." Attempts to establish places for isolation meet with much opposition and the solution projected is the formation of " colonies," the proposed constitution of which is given. Other measures in view are leper maternity work, the care of the children of lepers, and dissemination of knowiedge of the disease.

H. Harold Scott.

ARAUJO (H. C. de Souza). Como se combate a lepra em S. Paulo. [**How Leprosy is dealt with in S. Paulo.**]—*Sciencia Med.* 1929. Apr. Vol. 7. No. 4. pp. 193–199.

In S. Paulo there is an inspectorate of leprosy, consisting of a Chief Inspector, an Assistant Inspector, two dermatologists, two bacteriologists, a pharmaceutical chemist, five special sanitary inspectors, ten laboratory assistants and eight visitors. Every effort is made to detect cases in an early stage and to undertake treatment as soon as diagnosis is confirmed. The main leprosarium is that of S. Angelo, at present with 535 patients isolated, but capable of maintaining 800. There are two others which are designed to serve definite zones. Figures are given to show the cost of upkeep.

H. Harold Scott.

NEFF (E. A.) & SNODGRASS (Robert J.). **Leprosy—an Unusual Case of Family Infection.**—*Jl. Trop. Med. & Hyg.* 1929. May 15. Vol. 32. No. 10. pp. 137–138.

The authors report an instance—a family of Indians in Fiji, in which a son contracted leprosy from a neighbouring friend, and from him his father, mother and five of his six brothers and sisters got the infection, and only one child who had left the household early escaped.

L. R.

LUENGO GARCIA (Fabian). Lepra.—Dos casos observados en la kabila de Guezaua (Xauen). [**Two Cases of Leprosy seen in Guezaua (Xauen).**]—*Medicina Países Cálidos.* Madrid. 1929. July. Vol. 2. No. 4. pp. 361–364. With 3 text figs.

The first was a case of mixed leprosy of 3 years' duration, the second a nodular case of 2 years' duration. Treatment with antileprol proved very successful, 3 capsules daily and 2 intravenous injections weekly, on which days no capsules were given. The ulcers cicatrized in 15 days, and the drug was well tolerated, except for a troublesome cough coming on five minutes after the injections and lasting for 8–10 minutes.

H. Harold Scott.

CASAZZA (Roberto). Nota clinica sulle eruzioni acute nei lebbrosi con particolare riguardo all'eritema nodoso lebbroso. [**Acute Eruptions in Leprosy, in Particular Leprous Erythema Nodosum.**]—*Giorn. Ital. Dermat. e Sifil.* 1929. June. Vol. 70. Year 64. No. 3. pp. 529–556. [53 refs.]

Two cases of leprosy are described fully, and a brief reference is made to a third case, in which the disease was manifested by eruptions resembling erythema nodosum. The first was a woman of 50 years who had contracted leprosy nine years before. Her case was peculiar in that she exhibited with remarkable regularity exacerbations of fever and fresh outbreaks of the rash at monthly intervals. Much improvement followed treatment with antileprol. In the third case the eruption shortly preceded death. The author discusses the possible pathogeny of this manifestation, but without coming to any definite conclusion.

H. Harold Scott.

HENDERSON (John M.). **The Depigmented Patch in Leprosy: a Clinical and Pathological Study.**—*Indian Jl. Med. Res.* 1929. July. Vol. 17. No. 1. pp. 33–39. With 12 figs. on 6 plates (1 coloured). [10 refs.]

This is a detailed and illustrated description of the naked eye and microscopical appearances of the depigmented patches so commonly met with in nerve forms of leprosy, from which the author concludes that the partial depigmentation is due to the mechanical action of the proliferated endothelial cells interfering with the blood and lymph supply to a particular area; the nerve endings are also involved, producing the sensory symptoms.

L. R.

SAMSON (Jose G.) & LARA (C. B.). **Persistence of Nasal Lesions in Leprosy.**—*Jl. Philippine Islands Med. Assoc.* 1929. June. Vol. 9. No. 6. pp. 201–209. [1 ref.]

This paper contains further interesting data illustrating the tendency in cases recovering under treatment for lepra bacilli to persist in the nose after the skin has cleared up. Thus in 200 well-developed cases of leprosy 75 per cent. were positive to bacilli in the nasal mucosa, most frequently in the nasal septum, where lesions are commonly found at the junction of the bony and cartilaginous portions. In 685 examinations by the "Negative Examining Committee" the nose was found positive in 20·6 per cent. and the skin in 12·5 per cent. with ten examinations, but the proportion of positives with an increased number of examinations steadily decreased in the case of the skin and increased in that of the nasal mucosa. Even nodules may persist in the nasal mucosa after the skin has cleared up, and in a small group of relapsing cases nasal lesions only were found four times as frequently as skin ones alone. The examination of the nasal mucosa is therefore most important in re-examination of paroled negative cases.

L. R.

i. HOPKINS (Ralph). **Bone Change in Leprosy.**—*Radiology.* 1928. Dec. Vol. 11. No. 6. pp. 471–472. With 4 figs.

ii. COCHRANE (Robert G.). **Leprous Lesions of the Scalp.** [Correspondence.]—*Lancet.* 1929. June 1. p. 1170.

iii. NEFF (E. A.) & SNODGRASS (Robert J.). **Involvement of the Hairy Scalp in Leprosy: a Report of Two Cases.**—*Jl. Trop. Med. & Hyg.* 1929. June 1. Vol. 32. No. 11. pp. 147–148. With 1 text fig.

iv. WILSON (R. M.). **Leprosy in a Peripheral Nerve.**—*China Med. Jl.* 1929. June. Vol. 43. No. 6. pp. 617–618.

v. MUIR (E.) & HENDERSON (John M.). **An Obscure Neurological Case simulating Early Leprosy.**—*Indian Med. Gaz.* 1929. July. Vol. 64. No. 7. pp. 386–387. With 1 text fig.

vi. NAPIER (L. E.) & HENDERSON (J. M.). **Three Cases of Combined Leprosy and Dermal Leishmaniasis.**—*Ibid.* Aug. Vol. 64. No. 8. pp. 446–448. With 4 text figs. [1 ref.] [Calcutta School of Trop. Med. & Hyg., Calcutta.]

vii. MARCHOUX (E.). La lèpre maculeuse des noirs. [**Macular Leprosy in Blacks.**]—*Bull. Soc. Française Dermat. et Syph.* 1929. June. No. 6. pp. 619–620.

i. A short illustrated paper describing the well-known atrophy of the finger and toe bones in leprosy.

ii. The author points out that although leprous lesions of the scalp are rare in India they are not infrequent in Japan, including extensive nodules.

iii. Two cases of leprous lesions of the scalp seen in Fiji are reported, one nodular and one maculo-anaesthetic.

iv. A case is reported in a girl of 19 with two elongated tumours over the left shoulder-blade, which on removal proved to be leprous-thickened nerves.

v. A case of localized loss of sensation on the right side of the face simulating leprosy, but which proved to be due to a neoplasm of the sphenoidal sinus.

vi. Although post-kala-azar dermal leishmaniasis is a somewhat rare condition in Calcutta, the author reports having seen three cases combined with leprosy lesions in one morning, so suggests that there is some etiological relationship between them.

vii. A case of macular leprosy in a negro is reported and the difficulty in its recognition by the inexperienced is pointed out.

L. R.

LOWE (John). **Leprous Reaction.**—*Indian Med. Gaz.* 1929. Aug. Vol. 64. No. 8. pp. 438–441.

The author deals with this difficult subject as the result of much experience. After enumerating the tissues affected, he agrees with other workers that few lepra bacilli are found in the reacting tissues, mostly short and beaded ones, a polynuclear leucocytosis is present and the sedimentation rate of the red corpuscles is increased. The reactions are more common in poorly fed sedentary persons and they may be induced by such diseases as malaria and other fevers or vaccination. They may occur without any apparent reason, but most frequently follow treatment, especially with iodides or thyroid extract, but are not common after hydnocarpus preparations. Their nature is next discussed and the theory that they are allergic is favoured. All are agreed that severe and prolonged reactions are harmful, but there is much difference of opinion regarding the effect of mild ones, which MUIR regards as temporary and beneficial, but the author has found that reactions following potassium iodide always have a bad effect on the general condition of the patient, and that nerve reactions often result in permanent damage ; so he concludes that the hydnocarpus preparations, especially the ethyl esters, which show least tendency to produce reactions, give the best results. He agrees with MUIR that injections of tartar emetic and of adrenalin are most useful in the treatment of reactions.

L. R.

COCHRANE (Robert G.). **The Place of Potassium Iodide in the Management of Leprosy.**—*Jl. Christian Med. Assoc. India.* 1929. May. 6 pp. [Leper Asylum, Bankura, Bengal.]

The author at the outset lays stress on the importance of the general resistance of the body to leprosy and he discusses the question whether reactions are harmful or not in view of the breaking down of leprosy foci which accompanies them, especially when induced by iodides. He concludes that if lepra fever leaves the patient ill and unable to work it is harmful. In strong patients who can take vigorous exercise and with few lepra bacilli the breaking down of a few foci may stimulate the defensive mechanism and do good, as in early skin cases classed by MUIR as B1 in whom the slight reactions can easily be controlled, but in feeble flabby patients harm may be done by the drug by weakening him further, and it is also very dangerous in advanced nodular B3 cases

with myriads of lepra bacilli. Similarly in strong early nerve cases iodides may be used cautiously, but many early nerve A1 cases do not benefit by it. Experience at Bankura has shown that lepers do not improve more rapidly on iodides, while many deteriorate and become weaker, so they should only be used in strong patients and should be stopped if any weakness occurs and should rarely be continued for long, since a single reaction resembling protein shock may be beneficial, but repeated reactions harmful. Again, in cases apparently cured it is generally better not to risk producing a flare up by large doses of iodides, but to leave well alone, and it is also doubtful policy to give large doses as a diagnostic measure in those who have been exposed to infection.

L. R.

STÉVENEL (L.). Le principe actif de l'huile de chaulmoogra. [**Active Principle of Chaulmoogra Oil.**]—*Bull. Soc. Path. Exot.* 1929. May 8. Vol. 22. No. 5. pp. 338–341.

Commercial chaulmoogra oil is reported to be toxic to frogs and lizards. The author found this property to reside in the hard tegument of the hydnocarpus seeds and not in the pulp, and he thinks this part is also most active therapeutically when dissolved in olive oil. This active substance he regards as a kind of phytosterine. [It will be recalled that E. MUIR found the ethyl esters of olive oil to be therapeutically active in leprosy.]

L. R.

NEFF (M. E. A.). **Ethyl Esters of** *Calophyllum bigator*. **A Preliminary Report on Results following their Use at the Central Leper Hospital, Makogai, Fiji.**—*Jl. Trop. Med. & Hyg.* 1929. Sept. 2. Vol. 32. No. 17. pp. 241–243.

*Calophyllum bigator* is a Fijian tree the nut of which contains fatty acids and the crude adulterated oil has for long been used locally as an embrocation. The author reports that he has made an ethyl ester of the pure oil by Muir's method, and has used it as a local application for pains and also as an injection. He found it less active in leprosy than the ethyl esters and sodium salts of hydnocarpus oil, but thinks that a combination of the latter with the new ethyl ester is somewhat more effective. Moreover, the new preparation is particularly effective in nerve cases, and especially in rapidly relieving nerve pains, examples being given of cessation of severe and prolonged pains within a few hours after its injection in doses of 0·5 to 6 cc. intramuscularly, while no untoward effects have been noted beyond some tenderness at the site of the injection during two days.

L. R.

KIRBY-SMITH (J. L.). **Gold Sodium Thiosulphate in Maculo-Anesthetic Leprosy.**—*Southern Med. Jl.* 1929. July. Vol. 22. No. 7. pp. 637–639. With 3 text figs.

A mild bacteria-negative leprous skin infection was treated by six intravenous injections of 100 mgm. of this gold preparation with rapid improvement, but on the patient being informed of the nature of her disease she disappeared so could not be followed up.

L. R.

PALDROCK (A.). Die $CO_2$ -Schnee- und Lopionbehandlung der Lepra. [**Carbonic Acid Snow and Lopion Treatment of Leprosy.**]—*Arch. f. Schiffs- u. Trop.-Hyg.* 1929. Sept. Vol. 33. No. 9. pp. 455–461.

The author reports further on the carbonic snow treatment combined with gold preparations (see this *Bulletin,* Vol. 25, p. 207). He finds that preparations 2950 and 2949 are powerful in aiding the breaking up of the leprosy bacillus, but the latter, called lopion, is alone suitable for use in leprosy. Lopion can be given in larger doses than solganal, but considerable loss of weight, loss of appetite and intestinal derangement followed its use. The blood changes were similar with both preparations and the lipase increased to a similar degree. With smaller doses of lopion he thinks they will be equally effective.

L. R.

TRUFFI (Giovanni). La applicazione spinali di raggi X nei lebbrosi e nei tabetici ed il loro meccanismo di azione. [**Spinal Application of X-Rays in Leprosy and Tabes, and their Mode of Action.**]—*Giorn. Ital. Dermat. e Sifil.* 1929. June. Vol. 70. Year 64. No. 3. pp. 573–584. [36 refs.]

The analgesic effects of X-rays have been known almost since their first introduction into medicine. The author employed them on two tabetics with lightning pains, and on eight lepers, two suffering from the nervous form, two nodular, and four mixed. He used from 20–30 per cent. of the "erythema dose," applying them over the sites of the roots to which the painful areas would be referable, and at varying intervals, 10 to 20 days, on four or five occasions. In some cases the pains were relieved after the first application, and in all after the second. The relief was not permanent; in the tabetics especially the pains tended to recur in 30–50 days, but renewed applications procured further relief. Not only the pain but other symptoms were alleviated; the ataxy of the tabetics was improved and the trophic lesions of the lepers. The question of the mode of action is considered, whether due to indirect effects on the inflammatory accumulations at the nerve-roots, the ganglia, or the vertebrae, or to direct action on the somatic nerves, but it is concluded that the results are ascribable to action on the sympathetic, analogous to the relief obtained by operation on these branches.

H. Harold Scott.

KERR (Isabel). **Notes on the Value of the Sedimentation Test in the Treatment of Leprosy.**—*Indian Med. Gaz.* 1929. May. Vol. 64. No. 5. pp. 247–249. [Victoria Leprosy Hosp., Dichpali, Nizam's Dominions.]

The use of this test in the treatment of 120 cases of leprosy has shown it to be of great practical value, Muir's simple technic being used. (This *Bulletin,* Vol. 25, p. 982.) A low sedimentation rate of 20 to 28 indicates good resistant power and that potassium iodide can be used safely in addition to hydnocarpates, but if the rate is high, such as 40 or more, resistance is low and even small doses of KI may prove very harmful, so only hydnocarpates and tonics should be used. During treatment a rise of the index may continue after the clinical signs of a reaction have passed, and until it has fallen no further reaction-provoking treatment should be used. Hydnocarpates may

act as a tonic and reduce the sedimentation time and increase the patient's resistance ; as long as this drug produces the slightest reaction no KI should be given, so the test furnishes a reliable guide to safe and beneficial treatment.

L. R.

WALKER (Ernest Linwood). **Some New Aspects of the Etiology and Endemiology of Leprosy.**—*Jl. Preventive Med.* 1929. May. Vol. 3. No. 3. pp. 167–195. With 15 coloured figs. on 1 plate. [42 refs.] [George Williams Hooper Foundation for Med. Research, Univ. of California, San Francisco.]

In this paper it is claimed that the leprosy bacillus has been cultivated and proved to be a soil actinomyces. It can best be summarized in the author's own words :—

" The confusion and doubt surrounding cultivation of the lepra bacterium are due largely to the pleomorphic and facultative acid-fast characters of this organism.

" The acid-sensitive or partly acid-fast coccoid, diphtheroid, and actinomycoid organisms, that have been cultivated repeatedly from leprosy, are different stages in the life-cycle of the same organism.

" Hansen's bacterium in leprous lesions is probably the tissue stage of this pleomorphic and facultative acid-fast lepra organism.

" This organism cultivable from leprosy belongs in the genus *Actinomyces*, as now constituted, and is most nearly related to the facultative acid-fast species, such as *Actinomyces asteroides* and *A. caprae*.

" The difficulty of cultivation of the lepra bacterium, the failure of experimental infections of man with leprous material, and many of the clinical and pathological peculiarities of the disease appear to be due, in part at least, to the fact that the majority of the lepra organisms in the tissues of lepers are dead.

" Actual proof of the identity of the actinomyces cultivable from leprosy with Hansen's bacterium in the tissues, like proof of the etiologic relation of Hansen's bacterium to leprosy, would depend upon the experimental reproduction of the disease in animals. Notwithstanding the absence of such proof, the evidence in support of both relations is convincing.

" The actinomyces of leprosy, like other pathogenic actinomyces, is a soil organism probably of wide but irregular distribution, and is only a facultative parasite.

" Leprosy probably is primarily an infection from the soil, presumably through wounds ; but contagion as a possible secondary mode of dissemination is not thereby excluded.

" The bearing of these conclusions on methods of control of leprosy is obvious. It may ultimately be found that protection from soil-infected wounds, and proper cleansing and disinfection of contracted wounds, are at least as important as the segregation and isolation of lepers."

L. R.

GIORDANO (Mario). Ricerche culturali in casi di lebbra. [**Culture Researches in Leprosy.**]—*Arch. Ital. Sci. Med. Colon.* 1929. Jan. 1. Vol. 10. No. 1. pp. 8–13. With 3 coloured figs. on 1 plate. English summary p. 13. [Inst. of Trop. Path., Univ., Bologna.]

The author claims to have obtained a culture of the lepra bacillus on the medium of Hohn from the blood of a leper rich in the parasites, and to have obtained vigorous subcultures of acid-fast bacilli in the form of a streptothrix.

L. R.

SILVEIRA (Fleury) & GOMES (J. M.). A reacção de Kahn na lepra. [**The Kahn Reaction in Leprosy.**]—*Bol. Inst. Hyg. de São Paulo.* 1929. No. 39. 7 pp.

The authors carried out the Kahn, the Wassermann and the Deycke-Gomes reactions on 499 sera of patients showing clinically signs of leprosy without those of other diseases such as syphilis. The first proved much inferior to the second, since WR was positive in 204, whereas the Kahn only in 75.

In more detail, the following expresses the comparative results in tabular form :—

| Form of Disease. | Kahn + | Wassermann + | No. of Cases. |
|---|---|---|---|
| Nervous ... ... | 7 | 20 | 79 |
| Maculo-anaesthetic | 6 | 10 | 34 |
| Early ... ... | 28 | 28 | 91 |
| Mixed ... ... | 30 | 121 | 265 |
| Nodular ... ... | 4 | 25 | 30 |
| Total ... ... | 75 | 204 | 499 |

H. Harold Scott.

SILVEIRA (Fleury) & GOMES (J. M.). A reacção de Kahn sem cholesterina na lepra. [**The Kahn Test without Cholesterin in Leprosy.**]—*Ann. Paulist. Med. e Cirurg.* 1929. May–June. Year 17. Vol. 20. No. 5–6. pp. 50–53.

Parallel tests were performed with the sera of 29 non-syphilitic leper patients by means of the Kahn without cholesterin and the Wassermann.

The results have been arranged as in the previous abstract and were as follows, showing that the test deprived of cholesterin loses much of its value, 7 only out of 15 of the mixed form giving a negative.

| Form of Disease. | No. | ++++ | | +++ | | ++ | | + | | — | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | K | W | K | W | K | W | K | W | K | W |
| Nervous ... ... | 2 | 0 | 1 | 0 | 0 | 1 | 1 | 0 | 0 | 1 | 0 |
| Maculo-anaesthetic | 7 | 0 | 1 | 0 | 0 | 4 | 3 | 1 | 1 | 2 | 2 |
| Early ... ... | 2 | 0 | 0 | 0 | 0 | 0 | 1 | 1 | 1 | 1 | 0 |
| Mixed ... ... | 15 | 0 | 0 | 3 | 6 | 1 | 3 | 4 | 0 | 7 | 6 |
| Nodular ... ... | 3 | 0 | 0 | 0 | 0 | 1 | 3 | 0 | 0 | 2 | 0 |
| Total ... ... | 29 | 0 | 2 | 3 | 6 | 7 | 11 | 6 | 2 | 13 | 8 |

H. Harold Scott.

WILSON (R. M.) & GILMER (W. P.). **Complement Fixation and Precipitation Test for Syphilis in Lepers.**—*China Med. Jl.* 1929. May. Vol. 43. No. 5. pp. 484–486.

These tests in 637 lepers are compared with those on clinic patients in a hospital, using the Noguchi technique and the Kahn test, and very similar percentages of reactions were obtained, the strongly positive reactions being 14 and 19 per cent. respectively. L. R.

SCHLOSSMANN (K.). Vergleichende Studien über Lipoidstoffe der Leprabazillen, Tuberkelbazillen und der tierischen Organe. [**Comparative Studies on Lipoids of Leprosy and Tubercle Bacilli.**]—*Ztschr. f. Immunitätsf. u. Experim. Therap.* 1929. Aug. 1. Vol. 62. No. 5/6. pp. 447–463. [54 refs.] [Bact. Inst., Univ., Dorpat, Estonia.]

The author holds that it is still doubtful whether Hansen's bacillus has been cultivated, nor have serological reactions yet made early diagnosis and treatment possible, so that improved serological methods are required. As the result of his studies he obtained organic solutions of the extractable lipoids of 13 lepra bacilli cultures and from tubercle bacilli. The lipoid materials thus obtained gave different chemical reactions, but they could not be differentiated by micro-chemical analysis. Complement fixation tests with 33 antigens were carried out with the blood sera of 65 sick and healthy persons, but they did not allow of the serum diagnosis of latent leprosy, and the results were not conclusive.

L. R.

PALDROCK (A.). Die Blutgruppen der Leprösen in Estland. [**Blood Groups among Lepers of Estonia.**]—*Arch. f. Schiffs- u. Trop.-Hyg.* 1929. Aug. Vol. 33. No. 8. pp. 440–446. [16 refs.]

Individual blood groups behave differently in the sexes and in relation to predisposition and the onset and course of the disease. Thus the O and B groups are more predisposed to leprosy ; women are more predisposed than men in the B group. In the O group the disease appears earlier in life and females tend to be anaesthetic and males to be tubercular cases, but in A group both forms appear in either sex in the later years of adolescence. In the B group the disease tends to occur in either sex later in life and the cases run a more rapid course, especially in females. Should these blood group determinations be confirmed in a larger number of cases the conclusions will become important.

L. R.

MARRAS (Antonio) & FRAU (Alberto). Sulla tossicità dell' urina leprosa. [**Toxicity of the Urine in Leprosy.**]—*Giorn. Ital. di Dermat. e Sifil.* 1928. Dec. Vol. 69 (Year 63). No. 6. pp. 1665–1707. With 3 folding tables. [31 refs.]

Using rabbits as their experimental animals the authors made numerous observations of the results on the pupils, pulse, temperature, nervous system, etc., of the injection of urine from leper patients, and carried out post-mortem examinations. The results are recorded in detail in the text and also in tabular form. Fourteen leprous individuals were taken, two of the nodular type, six anaesthetic, five mixed, and one incipient case. Their findings may be summarized by stating that the toxicity proved to be lower than that recorded by those who have worked with normal urine. This held good regardless of the type of disease, of the period (whether during an exacerbation or a remission), and of the treatment. They maintain that though the bacillus gives rise to toxin and though there are renal changes found in leprosy, the function of urinary secretion is not interfered with and elimination of the toxin takes place by some other means, notably the skin and the intestine. The production of toxic substances in leprosy

is a slow process so that the organism has time to deal with them by these other routes. They do not exclude the possibility that changes in the vegetative nervous system may play a part, perhaps a large part, directly and indirectly in metabolism, and so bring about the hypotoxicity of the urine. This aspect of the question is to be further studied.

H. Harold Scott.

PETROV (V. P.). [**On the Leprosy and the Care taken of the Lepers in the Surhan-Daria District of the Usbekistan.**]—*Pensée Méd. d' Usbékistane.* Tashkent. 1929. No. 5. pp. 19–37. With 2 text figs. [160 refs.] [In Russian. English summary pp. 99–100.]

The author states that leprosy is spreading widely in Central Asia, but there are no reliable statistics of the disease and vitiligo is confused with it.

L. R.

SATOW (Tadashi). **Distribution of Lepers and its Prevention in Japan.**—*Jl. Public Health Assoc. Japan.* 1929. June. Vol. 5. No. 6. pp. 1–12.

This paper deals with the same figures as in the review in this *Bulletin, ante*, p. 610. The provision of leper hospitals in Japan is described.

L. R.

ROFFO (A. H.). Sobre la transmission de la lepra a los monos inferiores. [**Transmission of Leprosy to Lower Monkeys.**]—Reprinted from *Rev. Med. Latino-Americana.* Buenos Aires. 1926. Nov. Vol. 12. No. 134. 7 pp. with 3 coloured plates. French summary pp. 7–8. [3 refs.]

The inoculation of monkeys with leprous material produces only local tubercles, without infection of the nasal mucous membrane or general lesions, with an incubation period of about 30 to 35 days.

L. R.

STEIN (A. A.). [Sur la morphologie de la lèpre viscérale.] [**The Morphology of Visceral Leprosy.**]—*Arch. Sci. Biol.* 1929. Vol. 29. No. 2. pp. 135–165. [1 page of refs.] [In Russian. French summary pp. 165–166.]

The author describes the microscopical lesions in leprosy and states that by staining with Sudan III and carbolic fuchsin he demonstrated drops of lipoid material in the lepra cells.

L. R.

ABAL (P.). [Estudios serologicos en la lepra. Reserva alcalina.] [**Alkaline Reserve in Leprosy.**]—*Bot. Tecn. Direc. Gen. Sanidad.* 1929. Apr. Vol. 4. No. 4. [Summarized in *Bull. Inst. Pasteur.* 1929. May 31. Vol. 27. No. 10. p. 435.]

The blood of 1 anaesthetic and 9 mixed cases of leprosy was examined and the alkaline reserve was found to be normal.

L. R.

FIGUEREDO (Norbert). **The Use of Fibrolysin in Leprosy.**—*Indian Med. Gaz.* 1929. Aug. Vol. 64. No. 8. pp. 426–429. [3 refs.]

The supposed effects of fibrolysin in dissolving fibrous tissue has been exploited in a number of cases of leprosy with completely negative results.

L. R.

FERREIRA (Isauro Costa). Etiologia e prophylaxia da lepra.—*Folha Med.* 1929. Sept. 5. Vol. 10. No. 25. pp. 308–311.

FERRERO (Michelangelo). Ricerche sulla lebbra.—*Arch. Ital. Sci. Med. Colon.* 1928. Nov. Vol. 9. No. 11. pp. 647–675. [50 refs.] [Inst. of Colon. Path., Univ., Bologna.]

GALAN (Miguel A.). Un nuevo tratamiento para curar la lepra.—*Repert. Med. y Cirug.* Bogota. 1928. May. Vol. 20. No. 5 (229). pp. 230–247.

HOFFMANN (W. H.) & RAMOS BAEZ (P.). Las infecciones latentes de la lepra.—Reprinted from *Jl. dos Clinicos.* Rio de Janeiro. 1928. Vol. 9. No. 21. 16 pp.

DA MATTA (Alfredo). Escorço historico da lepra no Estado do Amazonas (1889 a 1928).—*Sciencia Med.* 1929. Apr. Vol. 7. No. 4. pp. 181–192. With 6 figs. on 4 plates .

DA MATTA (Alfredo). Os symptomas iniciaes das leproses. Seu contagio e evolução relativamente rapida.—*Sciencia Med.* 1929. May. Vol. 7. No. 5 pp. 233–249. With 5 figs. on 2 plates.

MISSRA (J. N.). O tratamento da lepra pelo succinol.—*Bol. Ger. Med. e Farmacia.* Bastora. 1929. Jan.–Apr. Ser. 13. No. 1-4. pp. 35–38.

SEMINARIO (C.) & GAVIÑA ALVARADO (E. R.). Lepra tuberculoide.—*Semana Méd.* 1929. May 30. Vol. 36. No. 22 (1846). pp. 1394–1396. With 1 text fig. [1 ref.]

DE SOUZA-ARAUJO (H. C.). Studies upon Leprosy. I. Transmission of Human Leprosy to the White Mouse. (Reprinted).—*Inst. Oswaldo Cruz. Supplemento das Memorias.* 1929. June 22. No. 8. pp. 123–127. With 7 figs. on 2 plates. [10 refs.] Reviewed in *T.D.B.* Vol. 26. p. 342.

## MISCELLANEOUS.

YEAGER (Clark H.). **Practical Bored-Hole Latrine Construction.** *Malayan Med. Jl.* 1929. June. Vol. 4. No. 2. pp. 45–55, With 18 text figs.

"Numerous requests have been received for information on the bored-hole latrine, many of which are for construction details. Therefore this article has been prepared to answer the questions which have been asked and to assist those persons who are unfamiliar with bored-hole latrine construction."

[Similar enquiries reach this Bureau and the article is therefore abstracted at some length. The illustrations are very poorly reproduced in the article but through the kindness of the Information Service of the Rockefeller Foundation it has been possible for the *Bulletin* to reproduce from original photographs.]

The general features of a bored-hole latrine are shown in Fig. 1.

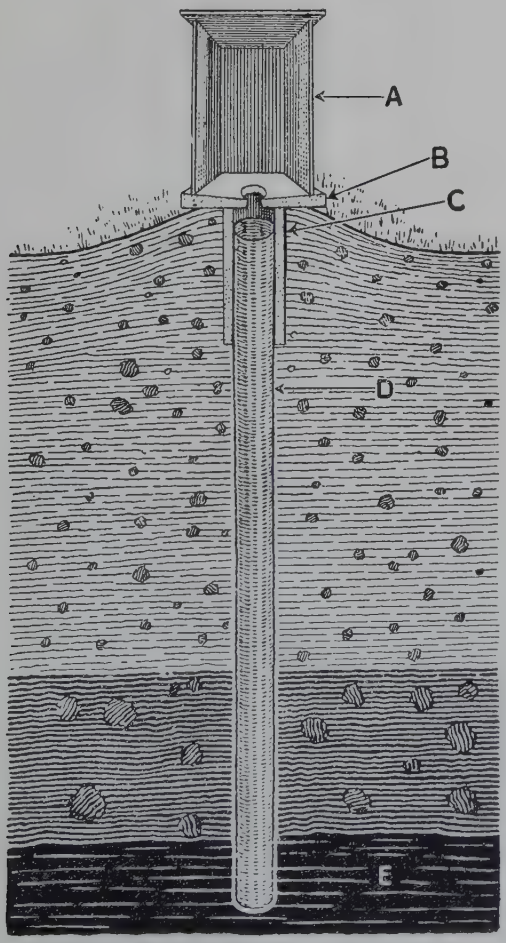


**Fig. No. 1.—THE TUBE LATRINE.**

A, Superstructure. B, Cement slab. C, Concrete cylinder, usually not necessary. D, Bamboo basket for retaining soft earth, not always necessary. E, Water.

The dimensions recommended for the hole are 20 feet deep and 16 in. diameter, i.e., a 16 in. auger is used. The latrines are most successful if the hole goes at least 3 ft. below the surface of the ground water.

*Requirements.*

(1) 16 in. auger-bottom (see fig. 4) which costs about 30s., landed in Malaya. This is best purchased separately as the shaft ordinarily supplied is not strong enough for use in latrine digging.

(2) Shaft for auger. For this is recommended a 22-feet length of $1\frac{1}{2}$ in. iron gas or water pipe, which is bolted to the auger-bottom and is drilled with $\frac{1}{2}$-in. holes at 18-in. intervals for attachment of crosspiece for turning. For convenience the shaft may be in sections (see fig. 2) using a coupling as illustrated.

(3) An ordinary iron "cross" or 4-way water pipe junction (see fig. 3). This is drilled and can be fixed at the desired position on the shaft by means of a bolt.

(4) Two 4 ft. turning arms of $1\frac{1}{2}$ in. iron pipe for attachment to the "cross" (see fig. 3).

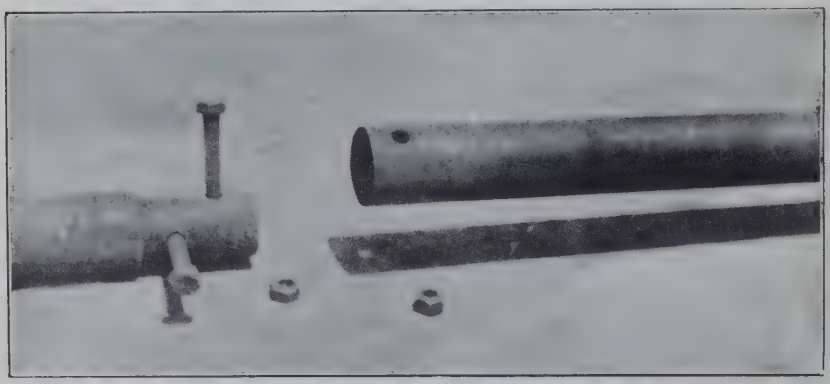

Fig. 2. Coupling for auger shaft.

Fig. 3. Four-way pipe junction with two turning arms.

(5) One tripod made from three 25-foot wooden poles or $1\frac{1}{2}$-in. pipe. This is for hauling up the auger full of soil (see fig. 5).

(6) One 12 in. pulley or gin block for hauling up auger. If necessary use a compound pulley.

(7) 50–60 feet of $\frac{3}{4}$-in. rope with hook on end. To engage the hook a ring can be fastened to the shaft at any convenient place.

(8) One pair of wooden hinged doors on a frame 36 in. square. In the free edge of each door is cut a semicircular notch. Once the auger has got down about 3 feet, the frame with its doors is placed flat over the hole so that when the doors are closed down, the shaft of the auger passes through the round hole made by the opposing notches. This steadies the auger during turning and makes it go down vertically.

(9) Twenty-foot length of 1 in. pipe with steel chisel riveted to the end. This is for breaking up soil too hard for the auger.

*Method of Use.*

The tripod is set up over the site chosen. The " cross " is slipped over the shaft. The rope running through the pulley is hooked to the shaft. The auger is erected to the boring position. If necessary cut a small hole by spade, removing roots, etc., to give the auger a start; this, however, is rarely necessary. Usually three turns fills the auger. Open doors, pull up auger, swing to side and empty (see fig. 5).

Sometimes large stones can be broken by the long tool mentioned in (9) above. Sometimes a small man must be lowered down to bring up the stone by his feet or a loop of rope. Pouring in water is helpful in some soils.

Fig. 4. Auger bottom.

Fig. 5. Tripod set up over chosen site for hauling up the auger full of soil.

*Difficulties.*

Sand sometimes requires the dumping of clay or mud into the hole to make the sand pack, or a lining may be pushed down as the work proceeds.

Rock would require special apparatus and the expense is generally not justified. If the rock is at 10 or 12 feet, make that the final depth.

Water may necessitate pumping or baling.

Special sand augers are obtainable. A lining of basket work made of split bamboo has been found useful to prevent caving (see fig. 1). Where the surface soil is soft an empty barrel or 18-in. cement cylinder will support the walls of the hole at the top.

If surface flooding is expected the cement cylinder should extend 2 feet above the highest water level.

*Floor of the latrine.*

A slab of reinforced concrete 30 by 36 by 2½ in. has been found satisfactory, sloping to a hole about 10 by 6 in. at the centre.

*Superstructure.*

This can be according to taste and local facilities.

*Variations of tools and methods.*

A number of these are suggested. For these the paper can be consulted, but many will occur to those putting the plan into practice.

J. F. C. H.

# INDEX OF AUTHORS OR SOURCES.

The bracketed abbreviations after the page numbers indicate the subjects. Page numbers within brackets indicate papers not summarized.

Am. signifies Amoebiasis and Amoebic Dysentery.
Bb. ,, Beriberi.
Bl. ,, Blackwater.
B.R. ,, Book Review.
Chl. ,, Cholera.
C.Bu. ,, Climatic Bubo.
Der. ,, Tropical Dermatology.
Dys. ,, Dysentery (Bacillary and Unclassed).
Ent. ,, Enteric Fevers.
Fev. ,, Fevers.
G.V. ,, Granuloma Venereum.
Hel. ,, Helminthiasis.
Hist. ,, Historical.
H.S. ,, Heat Stroke.
Jaun. ,, Infectious Jaundice.
K.A. ,, Kala Azar.
Lab. signifies Laboratory Reports.
Lep. ,, Leprosy.
Mal. ,, Malaria.
Misc. ,, Miscellaneous.
Myc. ,, Tropical Mycology.
Oph. ,, Tropical Ophthalmology.
Pel. ,, Pellagra.
Pl. ,, Plague.
Rab. ,, Rabies.
R.F. ,, Relapsing Fever and other Spirochaetoses.
Sp. ,, Sprue.
S.S. ,, Sleeping Sickness.
Tb. ,, Tuberculosis.
Und. ,, Undulant and Abortus Fever.
Y.F. ,, Yellow Fever.
Y. & S. ,, Yaws & Syphilis.
Z. ,, Medical Zoology.

## A


Aalsmeer, W. C. & Wenckebach, K. F., 517 (B.R.)
——, with ——, 352 (Bb.)
Abadia, G., with Bejarano & Rey, (37) (Mal.)
Abal, P., 1043 (Lep.)
Abd el Kader el Sabbagh, 871 (Am.)
Abell, E., with Huddleson, 73 (Und.)
Abella, R., with Vidal, 447 (Und.)
Aben-Athar, J., 343 (Lep.)
Abercrombie, T. F., 919 (Mal.)
Abuladse, S., 977 (Hel.)
——, with Kandelaki & Okropiridse, (949) (Mal.)
Acheson, J. A., 852 (Y. & S.)
Achundow, I., 250, 814 (Z.), 375 (Mal.)
Acton, H. W., 438 (B.R.)
—— & Knowles, R., 77 (B.R.)
—— & McGuire, C., 682 (Der.)
Adamantiadis, B., 506 (Oph.)
Adams, P., 716 (S.S.)
Adcock, E. W., 174 (Y. & S.), 653 (R.F.)
Adler, S., 325 (K.A.)
—— & Theodor, O., 256 (Z.), 324, 748, 749, 756 (K.A.)
Adowa, A. N. & Smirnowa, A. M., 814 (Z.)
——, with Smorodinzew, 814 (Z.)
——, with —— & Sébenzow, 812 (Z.)
Advier, M., (101) (Pl.)
Agramonte, A., 289 (Hist.)
Ahmad, B., with Sinton & Orr, 381 (Mal.)
Akazawa, S., 213 (S.S.)
Akowbjanz, G., 926 (Mal.)
Albernaz, P. M., 326 (K.A.)
Albuquerque, M. J. C., (862) (Y. & S.)
Alcock, A., 519 (Z.)
Aleixo, A., (344) (Lep.)
Alessandrini, A., 223 (Rab.)
—— & Pampana, E. J., 574, 575 (Misc.)
Alexa, I., with Ciuca, 24 (Mal.)
Alexeieff, A. & Gwéléssiany, J., 797 (Z.)
Allan, W., 91 (Pl.)
Allbutt, C., (291) (Hist.)
de Almeida, E., 709, 722 (S.S.), 866 (G.V.)
de Almeida, F. P., 764 (Myc.)
Alonso Alonso, L. & González Azcune, F., (949) (Mal.)
Alport, A. C., 564 (Misc.)
Alschibaja, K., 962 (Pel.)
Alvarez-Cienfuegos, J. M., 380 (Mal.)
Aly, M., with Shousha, 524 (Z.)
Amaret of Trans-Jordan Ann. Rep. Dept. Public Health, 6 (Mal.)
d'Amato, L., 596 (Misc.)
American Journal of Public Health, 37 (Mal.)
Anazawa, K., 249 (Z.)
Anderson, C., with Nicolle, 116, 118, 652, 659, 668 (668) *bis* (R.F.), 740 (K.A.)
——, with —— & Colas-Belcour, 118, 658, 668 (R.F.)
——, with —— & Hornus, 657 (R.F.)
——, with —— & Mathis, (128) (R.F.)
——, with —— & Rahal, 657 (R.F.)
——, with Wollman & Colas-Belcour, 242 (Z.)
Anderson, T. E., 118 (R.F.)
——, with Delorme, 126 (R.F.)
——, with Levaditi, 662 (R.F.)
——, with —— & Manin, 579 (Misc.)
Anderson, T. F., 907 (Mal.)
Ando, K., (632) (Chl.)
——, with Matsumoto & Shiraiwa, 87 (Chl.)
—— & Nishibe, M., 217 (Rab.)
André, with Labernadie, 341 (Lep.)
Angel Marín, R., 787 (Z.)
Angolotti, E., 330 (K.A.)
Anjow, S., 129 (R.F.)
Annesley, F. D., 572 (Misc.)
Antoine, E., 491 (Sp.)

Antonelli, G., (949) (Mal.)
Aoki, K., with Matsumura et al, 965 (Bb.)
Aragão, H. de B., 303, 305, 308, 650, 1001 *bis*, 1002, 1008 (Y.F.)
—— & Lima, A. da C., 1000 *bis*, 1001 (Y.F.)
Aramaki, Y., 175 (Y. & S.)
Araujo, H. C. de S., 342 *bis*, 609, 620 *bis*, 1032, 1035 (Lep.)
Araujo, O. da S., 169, 858 (Y. & S.), 184 (C.Bu.), 345 (B.R.)
Aravantinos, A., 1020 (Bl.)
Aravantinos, J., 836 (Fev.)
Arce, J., Balado, M. & Franke, E., (557) (Hel.)
——, Dimitri, V. & Balado, M., (557) (Hel.)
Archives des Instituts Pasteur d'Indochine, 401, 402 (Lab.)
Archives de l'Institut Pasteur de Tunis, 652 (R.F.)
Arellano, J. M., 26 (Mal.)
Arena, G., 11 (Mal.)
Arias Schreiber, L. & Escudero Franco, F., (824) (Z.)
Aristowsky, W. M. & Hoeltzer, R. R., 660 (R.F.)
—— & Schaechter, E. P., 127 (R.F.)
—— & Wainstein, A. B., 656 (R.F.)
Arnell, O. R., 1029 (Bl.)
Arneth, 562 (Misc.)
Artamonoff, A. S., (330) (K.A.)
Artamonow, A. S., 751 (K.A.)
d'Arusmond, L. P., with Legendre, (600) (Misc.)
Ascoli, M. & Sanfilippo, E., 68, 830 (Und.)
Ashby, C. F., 376 (Mal.)
Ashford, B. K., 493, 494 (Sp.)
Ashner, M., with Kligler, 44 (Fev.)
Askanazy, 986 (Hel.)
Assam, 222 (Rab.)
de Assis, A., 157, (165) (Dys.)
—— & Mendes, N. O., 156 (Dys.)
de Assumpção, L., 780 (Z.)
Aubertin, C. & Poumailloux, M., 671 (R.F.)
Aubrey, G. E., 675 (Fev.)
Audibert, 84 (Chl.)
Audry, C. & Viev, 624 (Lep.)
Augustine, D. L., Nazmi, M., Helmy, M. & McGavran, E. G., 553 (Hel.)
——, with Stuart, 542 (Hel.)
Aujeszky, A., 221, 225 (Rab.)
Auricchio, L., (489) (Dys.)
Avery, S. D., 591 (Misc.)
de Azevedo, A. P., 13 (Mal.)
——, with Torres, 299 (Y.F.)

## B

Baars, G., with Miessner, 225 (Rab.)
Bablet, J., 848 (Fev.)
Bacchelli, G., 202 (S.S.), 382 (Mal.), 789 (Z.)
——, with Franchini, 993 (Hel.)
—— & Taddia, (258) (Z.)
Bachman, G. W., 551 (Hel.)
Bachmann, N., 896 (Dys.)
Bacigalupo, J., 977 (Hel.)
Badger, L. F., with Wayson, 337 (Lep.)
Baer, J. G., with Joyeux, 975, 976 (Hel.)
Baermann, G. & Smits, E., 372 (Mal.)
Bailly, J., 221, 225 (Rab.)
Bailly, with Remlinger, 218, 222, 224, 736 *bis*, 737 *bis* (Rab.)
Baily, J. D., with Young, 362 (Mal.)
Bair, M. Z., 919 (Mal.)
Baker, C. E., with Stiles, 557 (Hel.)
Baker, J. R., 416 (Misc.)
Bakker, C., 505 (Oph.)
Bakunin, L., 191, 193, (724) (S.S.)
Balado, M., with Arce & Dimitri, (557) (Hel.)
——, with —— & Franke, (557) (Hel.)
Balaschewa, M. T., with Marzinowsky & Pickoul, 373 (Mal.)
Balfour, A., 414, 583 (Misc.), 857 (Y. & S.)
Balfour, M. C., with Carley, 919 (Mal.)
Balina, P. L., 335 (Lep.)
Ballif, L., with Ciuca & Viéru, 928 *bis*, 944 (Mal.)
—— & Gherscovici, I., 358 (Pel.)
—— & Lunevski, 381 (Mal.)
Bancroft, P., with Tomlinson, 765 (Myc.)
Banerjee, K. G., 14 (Mal.), 967 (Bb.)
Banerji, B. K., 569 (Misc.)
Barber, M. A. & Komp, W. H. W., 15 (Mal.)
——, —— & King, C. H., 921 (Mal.)
——, —— & Newman, B. M., 939 (Mal.)
Barbier, G., (625) (Lep.)
Barbieri, A., 247 (Z.)
Barbosa, J. P., (1008) (Y.F.)
Bardenwerper, H. E., 550 (Hel.)
Barg, I., with Bergman & Breyter, 788 (Z.)
Barlovatz, A., 573 (Misc.), 708 (S.S.), 853 (Y. & S.)
Baroni, with Scuderi, 880 (Am.)
Baroni, B., 758 (Myc.)
Barraud, P. J., 254, (258) (Z.)
——, with Sinton, 255 (Z.)
Barret, H. P., with Smith, 231 (Z.)
Barreto, J. de B., 1008 (Y.F.)
—— & Peryassú, A. G., 650 (Y.F.)
Barrett, J. W., 414 (Misc.)
Barron, M., 975 (Hel.)
Barros, S., 800 (Z.)
Barroso, S., (309 *ter*) (1008) (Y.F.)
Barrow, J. V., 794 (Z.)
Bass, E., 556 (Hel.)
von Bassewitz, B. E., (344 *bis*) (Lep.)
von Bassewitz, E., 108 (Der.)
Bastai, P., (74) (Und.)
—— & Rotta, C., 68 (Und.)
Basu, B. C., 809 (Z.)
Battistini, T. S., 850 (Fev.)
Bauer, J. H. & Hudson, N. P., 303 (Y.F.)
——, with —— & Philip, 647 (Y.F.)
Baumann, E. D., 287 (Hist.)
Baylis, H. A., 432 (B.R.)
Beacom, D. M., 561 (Misc.)
Bedford, G. A. H., 805 (Z.)
Beihefte zum Archiv für Schiffs- und Tropen-Hygiene, 876 (Am.)
Bejarano, J., Rey, G. V. & Abadia, G., (37) (Mal.)
Beklemischev, W. & Mitrophanova, J., 251 (Z.)
Bellgard, S. J., 1022 (Bl.)
Benarroch, E., 366 (Mal.)
——, with Hill, 371 (Mal.)
Benedetti-Valentini, F., (902) (Dys.)
Benedetto, de L., 1030 (Bl.)
Bensted, H. J., with Perry, 897 (Dys.), 973 (Hel.)

Bequaert, M., with Walravens & Valcke, 20, 931 (Mal.)
Berdnikov, V., 638 (Pl.)
Bérézнyack, M. E., 330 (K.A.)
Berger, E., (842) (Fev.)
Berglund, H., with Yang, 937 (Mal.)
Bergman, A., Breyter, A. & Barg, I., 788 (Z.)
Bergsma, S., 114 (R.F.)
Berman, P. & Leake, W. H., 565 (Misc.)
Bernard, P. N., (599) (Misc.)
Bernd, M., 893 (Dys.)
Bertwistle, A. P., 244 (Z.)
Bessemans, A. & Nélis, P., 133 (R.F.)
—— & Thiry, U., 671 (R.F.)
Bethea, O. W., 470 (Am.)
Bethoux, L., 828 (Und.)
Bevis, W. M., (625) (Lep.)
Bezsonova, A., 637 (Pl.)
Bharadwaj, A. C., (180) (Y. & S.)
Bhattacharji, S. P., with Megaw & Paul, 353 (Bb.)
Bhattacharyya, P. & Chowdhury, S. P. R., 372 (Mal.)
Bicalho, N., with Fialho & Pacheco, 300 (Y.F.)
Bichkov, V. & Borzenkov, A., 637 (Pl.)
Bideau with Dargein, 671 (R.F.)
Bidegaray, H., (258) (Z.)
Bigger, W. K., 6 )Mal.)
Biginelli, P., 378 (Mal.)
Bigot, A., Eyraud, R. & Velu, H., 736 (Rab.)
——, with Velu & Eyraud, 737 (Rab.)
Bilbao, L., with Figuerido, 7 (Mal.)
Billimoria, H. S., with Fairley & Mackie, 419 (Sp.)
Bird, W., 10, (387) (Mal.)
——, with Sinton, 14, 929 (Mal.)
——, with —— & Eate, 379 (Mal.)
——, with —— & Orr, 380 (Mal.)
Bishop, A., with Laidlaw & Dobell, 469 (Am).
Bishop, F. L., with Meleney & Roberts, 919 (Mal.)
Bisset, W., 743 (K.A.)
Blackmore, H. S., 531 (Hel.)
Blake, A. V. & Okell, C. C., 482, 889 (Dys.)
Blanc, G., (842) (Fev.)
—— & Caminopetros, J., 158 (Dys.), 327 (K.A.), 453, 833, 838, 839, 840 (Fev.)
——, ——, Dumas, J. & Saenz, A., 838 (Fev.)
——, —— & Giroud, P., 839 (Fev.)
——, —— & Joannidès, G., 761 (Myc.)
——, —— & Manoussakis, E., 42 (Fev.)
Blanchard, M. & Pin, M., 948 (Mal.)
——, with Toullec, 829 (Und.), 882 (Am.)
Bligh-Peacock, N., 525 (Hel.)
Bloch, B., 677 (Der.)
Bloch, J. I., (757) (K.A.)
Bloomfield, A. L. & Wyckoff, H. A., 496 (Sp.)
Blow, T. B., 246 (Z.)
Blumenthal, G., 541 (Hel.)
Blumenthal, W., 562 (Misc.)
Boak, R. & Carpenter, C. M., 448 (Und.)
——, with ——, 58, 73 (Und.)
Boase, A. J., 852, (862) (Y. & S.)
Bobes, S., (Rab.)
Boeck, W. C., 144 (Am.)
de Boer, H. S., 35 (Mal.)
Boettiger, C. & Werne, J., 986 (Hel.)
Bogoyavlensky, N. A., 918 (Mal.)
—— & Yampolskaya, (37) (Mal.)
Bohls, S. W., 728 (Rab.)
Boinet, Pieri, J. & Dunan, 49, 459 *bis* (Fev.)
Boletín del Instituto de Clínica Quirúrgica, Buenos Aires, 78 (B.R.), 990 (Hel.)
Boletin Oficina Sanitaria Panamericana, 101 (Pl.)
Bombay, 398 (Lab.)
Bonalberti, E., with Ottolenghi & Brighenti, 913 (Mal.)
Boncinelli, U., with Favilli, 72 (Und.)
Bonne, C., 762 (Myc.)
—— & Roskott, E. R. A. L., 927 (Mal.)
Bonnell, H. E., with Shaxby, 576 (Misc.)
Bonne-Wepster, J., with Walch, 806 *ter* (Z.)
Bonura, P., (387) (Mal.)
Boquet, A., 275 (Z.)
—— & Dujardin-Beaumetz, E., 639 (Pl.)
Borchardt, W., 986 (Hel.)
Borel, E., 248, 805 (Z.)
Borel, M., 5 (Mal.)
Borzenkov, A., with Bichkov, 637 (Pl.)
Bose, J. P., 426 (B.R.)
Bose, K., 909 (Mal.)
Botteri, J. H., 541 (Hel.)
Bouffard, G. & Legac, P., 818 (Z.)
Boughton, I. B., 736 (Rab.)
Boulay, A., Lhuerre, L. & Mitard, L., 24 (Mal.)
Bourgeois & Tsatsaronis, 339 (Lep.)
Bourriau, P., 475 (Am.)
Bourret, M., with Vigne, 752 (K.A.)
Boycott, A. E., 215 (Rab.), 804 (Z.)
Boyd, G. H., 802 (Z.)
Boyd, M. F., 810 (Z.)
Boyd, T. C. & Roy, A. C., 330 (K.A.)
Brahmachari, U. & Sen, P. B., 559 (Misc.)
Braun, H. & Weil, A. J., 479 (Dys.)
Breder, Jr., C. M., with Gudger, 791 (Z.)
Breindl, V., 800 (Z.)
Breyter, A., with Bergman & Barg, 788 (Z.)
Brighenti, D., with Ottolenghi & Bonalberti, 913 (Mal.)
Broden, A., 22 (Mal.), 149 (Am.)
Brooks, S. C., with Enlows, 484 (Dys.)
Broquet, C., 427 (B.R.)
Brosius, O. T., 15 (Mal.)
Brotzu, G., 71 (Und.)
——, with Ottolenghi, 923, 948 (Mal.)
Brown, H. W., 548 (Hel.)
Brown, P. W., 148 (Am.), (165) (Dys.)
Bruchmann, C. A. & de Nucci, L. S., 487 (Dys.)
Brug, S. L., 253, 812 (Z.), 546 (Hel.)
Brugi, A., 449 (Und.)
Brulé, Laporte & Ragu, 880 (Am.)
Brumpt, E., 230, 524 (Z.), 532 (Hel.)
—— & Neveu-Lemaire, M., 434 (B.R.)
Brun, G., 548 (Hel.)
Brunè, G. (258) (Z.)
Brunnekreeft, W. H. & Ongkiehong, H. F., 665 (R.F.)
Bruynoghe, R. & Dubois, A., 115 (R.F.)
Buchanan, G. S., 645 (Y.F.)
Buchanan, R. E. & Fulmer, E. I., 82 (B.R.)
Buchholz, L., (902) (Dys.)
Buchmann, M., 823 (Z.)
de Buck, A., Schoute, E. & Swellengrebel, N. H., (824) (Z.), 915 (Mal.)
Buckley, J. J. C., with Butler, 530 (Hel.)
Buddle, R., 242 (Z.)
de Buen, S., 19, (37), 374 (Mal.), 653 (R.F.)
——, with Casas & Rodriguez, 314 (K.A.)

Bulletin of the Antivenin Institute of America, 268, 270, 779 (Z.)
Bulletin de la Société de Pathologie Exotique, 170 (Y. & S.)
Bulletins from the Institute for Medical Research, Federated Malay States, 626 (Chl.)
von Bülow, T., 852 (Y. & S.)
Bunau-Varilla, P., 308 (Y.F.)
Bureau, Y., with Jeanselme & Giraudeau, 614 (Lep.)
Burke, A. M. B., 545 (Hel.)
——, with Hoffman & Marín, 544 (Hel.)
Burke, A. W., with Davis, 999 (Y.F.)
Burke, G. R., with O'Connor, 983 (Hel.)
Burnet, E., 67, 69, 443, 444 (Und.)
—— & Conseil, E., 829 (Und.)
—— & Durand, P., 844 (Fev.)
——, ——, & Olmer, D., 460 *bis* (Fev.)
—— & Olmer, D., 49 (Fev.)
Burnier, with Jeanselme & Eliascheff, 856 (Y. & S.)
Burnier, J. P., 508 (Oph.)
Burowa, L., 323 (K.A.)
Buschke, A. & Joseph, A., 104 (Der.)
Buss, G., 328, 747 (K.A.)
Busson, B., 216 (Rab.)
Butka, H. E., 792 (Z.)
Butler, C. S., 177 (Y. & S.), 289 (Hist.)
Butler, G. G., 391 (Lab.)
Butler, J. B. & Buckley, J. J. C., 530 (Hel.)
Butz, L. W., with Githens, 779 (Z.)
Buxton, P. A. (258) (Z.), 436 (B.R.)

## C

Cabanès, S. 346 (B.R.)
Caffrey, P. J., 488 (Dys.)
Calalb, G., 154 (Dys.)
——, with Jonesco-Mihaiesti, Combiesco & Condrea, 156 (Dys.)
Calcutta, 438 (B.R.)
Calderini, M., 726 (Rab.)
Calderón Blanco, S., (37) (Mal.)
Callewaert-Watsa, 686 (Der.)
Calvo Criado, V., 41 (Fev.)
Camacho, L., (599) (Misc.)
Cambessédès, H. & Cochez, 64 (Und.)
—— & Westphal, F., 1024 (Bl.)
Cameron, T. W. M., 533 (Hel.), 789 (Z.)
Caminopetros, J., with Blanc, 158 (Dys.), 327 (K.A.), 453, 833, 838, 839, 840 (Fev.)
——, with ——, Dumas & Saenz, 838 (Fev.)
——, with —— & Giroud, 839 (Fev.)
——, with —— & Joannidès, 761 (Myc.)
——, with —— & Manoussakis, 42 (Fev.)
Campbell, J. M., 907 (Mal.)
Campbell, M. F., 181 (G.V.)
Canal Feijóo, E. J., with Raimondi, 121 (R.F.)
Canning, G. A., 549 (Hel.)
Cannon, P. R. & McClelland, P. H., (524) *bis* (Z.)
Capéran, G., with Le Boucher, Laigret & Tchernenko, 997 (Y.F.)
Carbonell, M. V. (309) (Y.F.)
Cardamatis, J. P., 833 (Fev.)
——, with Savas, 428 (B.R.)
Carini, A., 238 (Z.)
Carley, P. S., 358 (Pel.)
—— & Balfour, M. C., (919) (Mal.)
Carmalt-Jones, D. W., 978 (Hel.)
Carman, J. A., 178 (Y. & S.)
Carpenter, C. M. & Boak, R., 58, 73 (Und.)
——, with ——, 448 (Und.)
Carpenter, G. D. H., 187 (S.S.)
Carrancá, R. & Trujillo, 963 (Pel.)
Carreno, C., with Maria Macera, (903) (Hel.)
Carri, M. A., with Fernández, (668) (R.F.)
Carrión, A. L., 264 (Z.)
Carroll, R. L., 23 (Mal.), 174 (Y. & S.), 271 (Z).
Carrosse, J. A. B. B. J., 120 (R.F.)
Carter, H. F., 241 (Z.)
—— & Jacocks, W. P., 947 (Mal.)
Cartron, 947 (Mal.)
Cartron, A., 841 (Fev.)
Carusi, R., 948 (Mal.)
Casas, U., de Buen, S. & Rodriguez, R., 314 (K.A.)
Casazza, R., 1035 (Lep.)
Castejon, R., 41 (Fev.)
Castellani, A., 102, 681, 684 (Der.), 155 (Dys.), 758, 761, 769, (769) (Myc.), (902) (Dys.)
—— & Duval, C. W., 689 (Der.)
—— & Mendelson, R. W., 866 (G.V.)
Castex, M. R. & Greenway, D., 232, (258) (Z.)
——, ——, Urdaniz, M. & Greenway, D. F., 476 (Am.)
Castigliola, O., (489) (Dys.)
Castronuova, G., (46) (Fev.)
Catanei, A., 103, 681, 682 (Der.)
——, with Foley & Giraud, 363 (Mal.)
——, with Montpellier & Cattoir, 769 (Myc.)
——, with Sergent, Edm. & Et., Parrot & Foley, 362, 363 (Mal.)
——, with ——, ——, ——, —— & Senevet, 3 (Mal.)
Cattoir, with Montpellier & Catanei, 769 (Myc.)
Cauchemez, L., 990 (Hel.)
Causeret, P. J. J., with Duguet & Escalier, (90) (Chl.)
Cavallini, E. & Crucillà, G., (387) (Mal.)
Cawston, F. G., 532 *bis*, 533 *ter* (Hel.), 687 *bis* (Der.), 790 (Z.)
Cazalas, X., 445 (Und.)
Cazanove, F., 643, 646, 995 (Y.F.)
Cazenave, E., 688 (Der.)
Celli, A., 283 (Hist.)
Celli-Fraentzel, A., 283 (Hist.)
Cerruti, C. F., 477 (Dys.)
Chagas, C., 308, 1002 (Y.F.)
Chagas, E., 299 (Y.F.)
—— & de Freitas, L., 996 (Y.F.)
Chaillot, L., with Normet & Urbain, 894 (Dys.)
Chamberlain, W. P., 783 (B.R.)
Chandler, A. C., 991 (Hel.)
——, with Chopra, 512 (B.R.)
Chaniotis, N. & Scordombekis, E., 836 (Fev.)
Charles, J. A., 153 (Dys.)
—— & Warren, S. H., 828 (Und.)
Chataway, J. H. H., 364 (Mal.)
Chatterjee, N. R., with Ghosh & Chopra, 569 (Misc.)
——, with Megaw & Ghosh, 23 (Mal.)
Chatterji, K. K., 875 (Am.)
Chatterji, S. P., with Henderson, 339 (Lep.)
Chaudhuri, S. G., with Chopra, 318 (K.A.)
Chester, W. L., 530 (Hel.)
Chesterman, C. C., 1022 (Bl.)
Chi, C. C., with Tsen, 735 (Rab.)

Chiabrera, G. & Reitani, U., 52 (Fev.)
Chiba, E., with Tanabe, 474 (Am.)
Chini, V., 727 (Rab.)
Chitre, G. D., with Mackie, 498, 499 (Sp.)
Chodukin, N. I. & Schewtschenko, F. I., 323 (K.A.)
Chodukine, N. J. & Soffieff, M. S., 324 (K.A.)
Chopra, R. N. & Chandler, A. C., 512 (B.R.)
—— & Chaudhuri, S. G., 318 (K.A.)
—— & Choudhury, S. G., 746 (K.A.)
——, David, J. C. & Dikshit, B. B., 567 (Misc.)
——, Dikshit, B. B. & David, J. C., 424 (Misc.)
——, with Ghosh & Chatterjee, 569 (Misc.)
——, —— & Dutt, A. T., 566 (Misc.)
——, Gupta, J. C. & Choudhury, S. G., 571 (Misc.)
——, —— & Ghosh, N. N., 566 (Misc.)
——, —— & Mullick, M. N., 171 (Y. & S.)
——, —— & Pillai, K. V., 564 (Misc.)
—— & Rao, S. S., 984 (Hel.)
Choudhury, B. K. P. & Morison, J., 901 (Dys.)
Choudhury, K. L., with Strickland & others 909 (Mal.)
Choudhury, S. G., with Chopra, 746 (K.A.)
——, with —— & Gupta, 571 (Misc.)
Chow, F. L., with Hindle, 910 (Mal.)
Chowdhury, K. L., 253, (258) (Z.)
——, with Strickland, Dodds-Price, Forsyth, Smith, Murphy & Williams, 4 (Mal.)
Chowdhury, S. P. R., with Bhattacharyya, 372 (Mal.)
Christophers, S. R., Sinton, J. A. & Covell, G., 360 (Mal.)
Christopherson, J. B., 984 (Hel.)
Chuan, L. T., with Tsuchiya, 634 (Pl.)
Chukerbuti, J. C., 563 (Misc.)
Ciccimarra, G., (668) (R.F.)
Cilento, R. W., 46 (Fev.), 585 (Misc.)
Ciuca, M. & Alexa, I., 24 (Mal.)
——, Ballif, L. & Viéru, M., 928 *bis*, 944 (Mal.)
Clark, H. C., 2 (Mal.), 271 *bis* (Z.)
Clark, O., (37) (Mal.)
Clarke, J. L., with Jackson, van Hovenberg, Filby & Green, 37 (Mal.)
Clarkson, L. M., 919 (Mal.)
Claude, 947 (Mal.)
Clayton, F. H. A. & Warren, S. H., 894 (Dys.)
Clearkin, P. A., 139 (Fev.)
Clemente, G., 767 (Myc.)
Cleveland, L. R., 233 *bis*, 234 (Z.)
Cluzel, 34 (Mal.)
Cochez, with Cambessédès, 64 (Und.)
——, with Meslay-Coirre, 330 (K.A.)
Cochrane, R. G., 1036, 1037 (Lep.)
——, with Crozier, 615 (Lep.)
—— & Mittra, P., 340 (Lep.)
Cock, F., 559 (Misc.)
Cohen, A. J., 547 (Hel.), 861 (Y. & S.)
Colas-Belcour, J., 822 (Z.)
——, with Nicolle & Anderson, 118, 658, 668 (R.F.)
——, with Wollman & Anderson, 242 (Z.)
Colby, S., with Hershfield, Kibler, Koenig, Schmid & Saunders, 675 (Fev.)
Cole, W. H. & Heideman, M. L., 874 (Am.)
Coleman, M. B., with Gilbert, 439 (Und.), 895 (Dys.)
Collart, A., with Schwetz & Geernick, 939 (Mal.)
Collignon, M., 1009 (Bl.)
Colonieu, L., with Raynard, 328 (K.A.)
Colwell, C., with Davis, 797 (Z.)
Comaroff, R., with Kligler, 715 (S.S.)
Combiesco, D., with Ionesco-Mihaiesti, Condrea & Calalb, 156 (Dys.)
Commonhealth, Mass., 226 (Rab.)
Compton, A., 639 (Pl.)
Comyn, K., (38) (Mal.)
Condrea, P., with Ionesco-Mihaiesti, Combiesco & Calalb, 156 (Dys.)
Connal, A., 390 (Lab.), 1015 (Bl.)
Connor, F. P., 430 (B.R.)
Conseil, E., 842 *bis* (Fev.)
——, with Burnet, 829 (Und.)
Coogle, C. P., 33 (Mal.)
Cook, A. R., 412 (Misc.), 859 (Y. & S.)
Cook, C., 347 (B.R.)
Cook, D. H. with Taliaferro & Hoffman, 534, 535 (Hel.)
Cooke, F. H., 334 (Lep.)
Cooke, W. E., with Low & Martin, 1023 (Bl.)
—— & Willoughby, H., 1021 (Bl.)
Cool, P., 122 (R.F.)
Cooley, R. A., 244 (Z.)
Coombs, F. S., 587 (Misc.)
Coonoor, Southern India, 735 (Rab.)
Coope, A. B. J., 424 (Misc.)
Copanaris, P., 40 (Fev.), 450 (Fev.)
Corcuff, C., 238 (Z.)
Cordes, W., 16 (Mal.), 171 (Y. & S.)
Corkill, B., (489) (Dys.)
——, with Penfold, 52 (Fev.)
Cormack, R. P., 1014 (Bl.)
Corpus, T., 626 (Chl.)
Corradi, A., 938 (Mal.)
Corson, J. F., 206, 697 (S.S.)
Cort, E. C., 164 (Dys.), 466 (Am.), 1017 (Bl.)
Cort, W. W., Otto, G. F. & Spindler, L. A., 985 (Hel.)
Cortes, N. A. F., 329 (K. A.), (388) (Mal.)
Costa, M. O., 502 (Sp.)
Costa-Mandry, O. 502 (Sp.)
—— & Marin, R. A., 466 (Am.)
Cousin, G., 816 *bis* (Z.)
Coutelen, F., 821 (Z.), 886 (Am.), 979 (Hel.)
Covell, G., 4, 5 (Mal.)
——, with Christophers & Sinton, 360 (Mal.)
Coventry, F. A., 549 (Hel.), 799 (Z.)
Covisa, J. S. & Gonzalez Medina, R., 315 (K.A.)
Cowdry, E. V. & Cowell, W. P., 239 (Z.)
—— & Scott, G. H., 239 (Z.)
Cowell, W. P., with Cowdry, 239 (Z.)
Craig, C. F., 143, 150 (Am.), 368 (Mal.)
Craighead, A. C., with Shortt, Smith & Swaminath, 322, 323 (K.A.)
——, with —— & Swaminath, 310 (K.A.)
Crawford, E. J., 174 (Y. & S.)
C.R. du Premier Congrès Antipesteux de l'U.R.S.S. Saratov du 31 mai au 3 juin 1927, 92 (Pl.)
Crozier, G. G., 1033 (Lep.)
—— & Cochrane, R. G., 615 (Lep.)
Crucillà, G., with Cavallini, (387) (Mal.)
Cruz, J. da C., 888 *bis* (Dys.), 1003 *bis* (Y.F.)
da Cruz, L. J. C., with de Mello, 475, 878 (Am.)

Cuatrecasas, J. & Garcia Tornel, L., (832) (Und.)
Cuenod & Nataf, R., 505 (Oph.)
da Cunha, A. M. & Muniz, J., 55, 463 (Fev), 295, 303, 308, 646 (Y.F.), (258) (Z.), (902) (Dys.)
Cunningham, J., 222 (Rab.), 517 (B.R.)
——, Nicholas, M. J. & Lahiri, B. N., 219 *ter* (Rab.)
Curran, J. A., 230 (Z.)
Curry, D. P., 821 (Z.)
Curson, H. H., 261, 786 (Z.)
Curtis, L. F., with Williams & Wildman, 869 (Am.)
van Cutsem, A., 882 (Am.)

## D

Dagnini, G. & Strozzi, T., (668) (R. F.)
Dake, W. J. L., 413 (Misc.)
Dalal, K. R., 968 (Bb.)
Dalrymple, J., 110 (Der.)
Danilow, B. A., 813 (Z.)
Dargein & Bideau, 671 (R.F.)
—— & Oudard, 469 (Am.)
Darré, H. & Laffaille, A., 446 (Und.)
Das, C., with Hodgson & Sen, 321 (K.A.)
Datta, S., 546 (Hel.)
Daukes, S. H., 953 (B.R.)
David, A., 757 (K.A.)
David, H., 224 (Rab.)
David, J. C., with Chopra & Dikshit, 424, 567 (Misc.)
Davies, W. M., 817 (Z.)
Dávila, J. V., with Phelps, 162 (Dys.)
Davis, C. H. & Colwell, C., 797 (Z.)
Davis, G. E., with Hudson & Philip, 1005 (Y.F.)
Davis, N. C., 252 (Z.), 1000 (Y.F.)
—— & Burke, A. W., 999 (Y.F.)
—— & Rickard, E. R., (949) (Mal.)
—— & Shannon, R. C., 253 (Z.), 1000 (Y.F.)
Davis, O., (902) (Dys.)
Davis, W. D., (902) (Dys.)
Davison, A. R., 615 (Lep.)
Dawley, C. W., with Miller, Jr., 540 (Hel.)
De, J. C., 47 (Fev.)
De, N. K., with Henderson & Ghosh, 617 (Lep.)
Dearman, W. A., 356 (Pel.)
De-Georges, L., 728 (Rab.)
De la Guardia, J., 760 (Myc.)
Delamare, G. & Gatti, C., 106, 683 *bis*, 684 (Der.), 667 (R.F.), 865 (G.V.)
—— & Giagni, 866 (G.V.)
Deland, C. M., 103 (Der.)
Delanoë, P., 823 (Z.)
De la Torre, T., 22 (Mal.)
De la Vega, J. F., 330 (K.A.)
Delbanco, E., Mühlens, P. & Vogel, H., 984 (Hel.)
Del Favero, E., (599) (Misc.)
Delorme, M. & Anderson, T. E., 126 (R.F.)
——, with Troisier, Deschiens & Limousin, 556 (Hel.)
Demina, N. A. & Nicolsky, W. W., 252 (Z.)
Denney, O. E., 608 (Lep.)
——, with Hopkins, 607 (Lep.)
Denys, P., 892 *bis* (Dys.)
Deschiens, R. & Melnotte, P., 465 (Am.)
——, with Troisier, Limousin & Delorme, 556 (Hel.)
Deutsch, B., (388) (Mal.)
Devi, A. L., 752, 753 (K.A.)
Dew, H., 980 (Hel.)
Dew, H. R., 691 (B.R.)
Dey, N. C., 751 (K.A.)
Dhar, D. R. & Sen, K. C., 87 (Chl.)
Díaz, N., (757) (K.A.
Díaz Flores, A., (949) (Mal.)
Dik, J. H., with Van den Hoven van Genderen 224 (Rab.)
Dikshit, B. B., with Chopra & David, 424, 567 (Misc.)
——, with Gupta, 752 (K.A.)
Dimancesco-Nicolau, O., with Nicolau & Galloway, 727 (Rab.)
Dimitri, V., with Arce & Balado, (557) (Hel.)
Dimow, D. K. & Nakhapetoff, M. I., 65 (Und.)
Dinger, J. E., Schüffner, W. A. P., Snijders, E. P. & Swellengrebel, N. H., 1006 (Y.F.)
Dionisio Gutierrez, P. & Hizon, J., 111 (Der.)
Dircks-Dilly, J., 987 (Hel.)
Dixey, M. B. D., 987 (Hel.)
Djaparidse, P., (949) (Mal.)
Djaparidze, P. S., 962 (Pel.)
Dobell, C., 795 (Z.)
——, with Laidlaw & Bishop, 469 (Am.)
Dodds-Price, J., with Strickland, Chowdhury, Forsyth, Smith, Murphy & Williams, 4 (Mal.)
Donatien, A. & Lestoquard, F., 755 (K.A.)
——, —— & Parrot, L., 755 (K.A.)
Donnison, C. P., 593 (Misc.), 1022 (Bl.)
Doorenbos, W., 480 (Dys.)
Doria, R., 447 (Und.)
Doubrow, with Garin & Mounier, 991 (Hel.)
Doubrowine, V. P., 651 (R.F.)
Doucas, C., 751 (K.A.)
Doughty, J. F., 273 (Z.)
Dove, W. E., with Kirby-Smith & White, (993) (Hel.)
Dowden, R., 360 (Mal.), 399 (Lab.)
Dowling, O., 919 (Mal.)
Draper, A. P., 23 (Mal.)
Drennsn, L. M., 147 (Am.)
Drenowsky, A. K., 370 (Mal.)
van Driel, B. M., 53 (Fev.)
Dschaparidse, P., 928 (Mal.), 987, 993 (Hel.)
Dubois, A., 209 (S.S.)
——, with Bruynoghe, 115 (R.F.)
Dubreuilh, W., 102 (Der.)
Duff, W. R., with M'Hutchison, 18 (Mal.)
Duffau & Lallement, 633 (Pl.)
Dugdale, J. N., 423 (Misc.)
Duguet, M. L. F., Causeret, P. J. J. & Escalier, J. A. E., (90) (Chl.)
Duhig, J. V. & Jones, G., 278, 279 (Z.)
Dujardin-Beaumetz, E., with Boquet, 639 (Pl.)
Duke, H. L., 204 (S.S.)
——, Hall, G. N. & Haddon, E. C., 262 (Z.)
Duke-Elder, W. S. & Duke-Elder, P. M., 509 (Oph.)
Dumas, J., with Blanc, Caminopetros & Saenz, 838 (Fev.)
Dunan, J., 844 (Fev.)
Dunan, with Boinet & Piéri, 49, 459 *bis* (Fev.)
Duncan, J. T., 441 (Und.)

Dunlop, G. A., 54, 845 (Fev.)
Duprey, A. B., 370 (Mal.)
Dupuy, H., 1026 (Bl.)
Durand, P., with Burnet, 844 (Fev.)
——, with —— & Olmer, 460 *bis* (Fev.)
Duren, (599) (Misc.)
Duren, A. N., 813 (Z.)
Dutt, A. T., with Chopra & Ghosh, 566 (Misc.)
Duval, C. W. with Castellani, 689 (Der.)
Duyster, M., 565 (Misc.)
Dyar, H. G., 514 (B.R.)

## E

Eate, S. N., with Sinton & Bird, 379 (Mal.)
Eber, C. T., 507 (Oph.)
Ebstein, E., 285 (Hist.)
Eggleston, C., with Hunt, McCann, Rowntree, & Voegtlin, 378 (Mal.)
Egypt, 509 *bis* (Oph.)
Ehara, I., 624 (Lep.)
Eisenmenger, C., with Feldt, 423 (Misc.)
Eisma, M., 554 (Hel.)
El Batash, M. H., 971 (Hel.)
——, with Khalil, Nazmi, Peter & El Din, 972 (Hel.)
El Din, M. S., with Khalil, Nazmi, Peter & El Betash, 972 (Hel.)
Eliascheff, with Jeanselme & Burnier, 856 (Y. & S.)
Eliot, C. P., with Ford, 524 (Z.)
Elkeles, G., 894 (Dys.)
Elkington, J. S. C., 583 (Misc.)
Elmes, B. G. T., with Smith, 688 (Der.)
Elvery, P. G. M., 583 (Misc.)
Embil, V. A., with Hoffmann, 791 (Z.)
Emery, F. E., with Herrick, 549 (Hel.)
Enciso, A., 3 (Mal.)
Enlows, E. M. A. & Brooks, S. C., 484 (Dys.)
Eristawi, K., (902) (Dys.), 948 (Mal.)
Ermilova, E. & Kruchkova, E., 579 (Misc.)
Escalar, G., with Pecori, 34 (Mal.)
Escalier, J. A. E., with Duguet & Causeret, (90) (Chl.)
Escomel, E., 750 (K.A.)
Escudero Franco, F., with Arias Schreiber, (824) (Z.)
Esquivel, R., with Lenci, 220 (Rab.)
Essed, W. F. R., 253 (Z.), 912 (Mal.)
Evans, W. H., 154 (Dys.)
Ewing, H. E., 957 (B.R.)
Eyl, M., with Richter & Maurer, 540 (Hel.)
Eyraud, R., with Bigot & Velu, 736 (Rab.)
Eyre, J., Notton, H. E. F. & Pope, W. J., 567 (Misc.)

## F

Facio, A. A., 497 (Sp.)
Fairley, K. D., 977 (Hel.)
Fairley, N. H., 770 *bis*, 775 (Z.)
——, with Mackie, 500 (Sp.)
——, —— & Billimoria, H. S., 491 (Sp.)
—— & Splatt, B., 773 (Z.)
—— & Wright-Smith, R. J., 979 (Hel.)
Fanelli, G., 476 (Am.)
Far Eastern Association of Tropical Medicine, 517 (B.R.)
Faust, E. C., 539, 970, 976 (Hel.), 786 *bis* (Z.)
Faust, E. C. & Kellogg, C. R., 786 (Z.)
——, with Li, 539 (Hel.)
——, with Zia, 29 (Mal.)
Favilli, G., (74) (Und.)
—— & Boncinelli, U., 72 (Und.)
Favre, with Nicolas & Lebeuf, 862 (C.Bu.)
Fawcett, J. & Gibson, A. G., 768 (Myc.)
Federated Malay States, 36, 360 (Mal.), 399 (Lab.)
Feemster, R. F. & Feemster, O. S., 563 (Misc.)
Feldt, A. & Eisenmenger, C., 423 (Misc.)
Feldt, A. M., 528 (Hel.)
Ferguson, E. W., 40 (Fev.)
Fermi, C., (38), 369 (Mal.), 245 (Z.), 730 *bis*, 731 *quat.* 733, 734 (Rab.)
—— & Lumbau, D., 729 (Rab.)
Fernandez, E. C., with Loganadan, 896 (Dys.)
Fernández, J. G. & Carri, M. A., (668) (R.F.)
Fernando, S. E., with Spittel, 690 (Der.)
Fernós Isern, A., 586 (Misc.)
Ferrari, A., (388) (Mal.), 560 (Misc.), 996 (Y.F.)
Ferreira, I. C., (1044) (Lep.)
Ferrer, H., 506 (Oph.)
Ferrero, M., (1044) (Lep.)
Ferruccio, C. R., (355) (Bb.), 858 (Y. & S.)
Fettweis, M., 899 (Dys.)
Fialho, A., 636 (Pl.), 998 (Y.F.)
——, Bicalho, N. & Pacheco, G., 300 (Y.F.)
——, with Jakob, Villela A. & E. L., 298 (Y.F.)
——, with de Mello, 123 (R.F.)
Fichera, S., 895 (Dys.)
Fiera, E. R., 851 (Y. & S.)
Fiertz, C. O., with Kopeloff, 30, 386 (Mal.)
Fiessinger, N. & Walter, H., 1028 (Bl.)
Fife, C. A., with de Rivas, 527 (Hel.), 787 (Z.)
de Figueiredo, B., with Penna, (1008) (Y.F.)
Figueredo, N., 1043 (Lep.)
Figuerido, C. A. & Bilbao, L., 7 (Mal.)
Filby, E. L., 919 (Mal.)
——, with Jackson, van Hovenberg, Green & Clarke, 37 (Mal.)
Finik, Z. & Mglej, S., 736 (Rab.)
Finney, W. P., 273 (Z.)
Finocchio, M., with Volpino, 221 (Rab.)
Fischer, O., 18, 945 (Mal.), 577 (Misc.), 878 (Am.)
—— & Rheindorf, G., 376 (Mal.)
Fischer, W., 529 (Hel.)
Fischl, F., 863 (C.Bu.)
Fischl, V., with Steiner, 653 (R.F.)
Flandin, C., Marchal, G. & Langlois, L., (949) (Mal.)
Fletcher, W., Lesslar, J. E. & Lewthwaite, R., 52, 848 (Fev.)
Flora, G., with Portocalis, 453 (Fev.)
Flu, P. C., 638 *bis* (Pl.)
Foley, H., Catanei, A. & Giraud, A., 363 (Mal.)
——, with Sergent, Edm. & Et., Parrot & Catanei, 362, 363 (Mal.)
——, with ——, ——, —— & Senevet, 3 (Mal.)
Fonquernie, J., 99 (Pl.)
da Fonseca, O., (309) (Y.F.), 679 (Der.)
—— & Leao, A. E. de A., 766 (Myc.)
Forbes, (949) (Mal.)
Ford, W. W. & Eliot, C. P., 524 (Z.)
Forbes-Brown, 878 (Am.)
Forsyth, C. E. P., with Strickland, Chowdhury, Dodds-Price, Smith, Murphy & Williams, 4 (Mal.)

Fort, M. A., 551 (Hel.)
Fouad, A. M. M., 971 (Hel.)
Fourche, J. A., 191 (S.S.)
—— & Ricklin, J., 193 (S.S.)
Fox, H., 105 (Der.)
Fraga, C., 294 *ter*, (309) (Y.F.)
Franchini, G., 240, 244 *bis*, 248, 266, 800 (Z.), (489) (Dys.), 342, 614, 625 (Lep.)
—— & Bacchelli, G., 993 (Hel.)
Franke, E., with Arce & Balado, (557) (Hel.)
Frankel, E., 485 (Dys.)
Franklin, E. M., 852 (Y. & S.)
Frasey, V., with Pettit & Stefanopoulo, 307 (Y.F.)
Frau, A., with Marras, 1042 (Lep.)
Frei, W. & Koppel, A., 861 (Y. & S.)
Freiman, M., 930 (Mal.)
de Freitas, L., with Chagas, 996 (Y.F.)
Freudenthal, P., (558) (Hel.)
Fried, S. M. & Segal, M. B., 679 (Der.)
Friedheim, E. A. H., 621 (Lep.)
Frobisher, Jr., M., 1004 (Y.F.)
Fróes, H., (388 *ter*) (Mal.)
Frugiuele, T., with Tegoni & Williams, (38) (Mal.)
Fuchs, D., 753 (K.A.)
Fujimori, K., 629, 630 (Chl.)
Fujinami, A., 528 (Hel.)
Fujisaki, K., with Matsumura et al, 965 (Bb.)
Fujisaki, T., 547 (Hel.)
Fujiwara, A., 623 (Lep.)
Fukushima, B., 123 *bis* (R.F.)
Fülleborn, F., 513 (B.R.)
Fullerton, H. R., 919 (Mal.)
Fulmer, E. I., with Buchanan, 82 (B.R.)
Furukawa, H., 212 (S.S.)
Fusco, G., (642) (Pl.)

## G

Gabbi, U., (842) (Fev.)
Gabriélidès, D., 504 (Oph.)
Gage, I. M., 866 (G.V.)
Galan, M. A., (1044) (Lep.)
Galbreath, W. R. & Weiss, C., 769 (Myc.)
Gallais, G., 12 (Mal.)
Galliard, H., 126 (R.F.), 723 (S.S.)
Galli-Valerio, 986 (Hel.)
Galloway, D., 759 (Myc.)
Galloway, I. A., with Nicolau & Dimancesco-Nicolau, 727 (Rab.)
Galt, C. M., 416 (Misc.)
da Gama, L. A., 562 (Misc.)
Gambier, A., (101) (Pl.)
Gaminara, A., 277 (Z.)
Ganguly, L. B., 627 (Chl.)
Garcia, O., 89 (Chl.)
Garcia Tornel, L., with Cuatrecasas, J., (832) (Und.)
Gardner, G., 131 (R.F.)
Gardner, L. L., with St. John & Simmons, 988 (Hel.)
Garin, C., Doubrow & Mounier, 991 (Hel.)
Garofali, F., (949) (Mal.)
Garton, W. M., 35 (Mal.)
Gasser, R. R., 180 (Y. & S.)
Gastaminza, U., 370 (Mal.)
Gaté, J. & Michel, P., 862 (C.Bu.)
Gater, B. A. R., 805 (Z.)
Gatti, C., with Delamare, 106, 683 *bis*, 684 (Der.), 667 (R.F.), 865 (G.V.)
Gaviña Alvarado, E. R., with Seminario, (1044) (Lep.)
Gavrila, I., 963 (Pel.)
Geernick, with Schwetz & Collart, 939 (Mal.)
Geiger, A., with Kligler, 718 (S.S.)
Geiger, J. C., with Williamson & Kaplan, 870 (Am.)
Gelonesi, G., 128 (R.F.) (489) (Dys.), 675 (Fev.)
Geneeskundig Tijdschrift voor Nederlandsch-Indië, 402, 403 (Lab.)
General Government of French West Africa, 602 (B.R.)
Genoese, G., 1025 (Bl.)
—— & Zallocco, A., 370 (Mal.)
Genovese, F., (949) (Mal.)
Geoghegan, A. J., 203, 704 (S.S.)
Georgopoulos, M., 453 (Fev.)
Gerlach, F. & Schweinburg, F., 726 (Rab.)
Gessner, F. E., 634 (Pl.)
Gessner, O., 877 (Am.)
Ghalib, A., with Hauduroy, 638 (Pl.)
Gherscovici, I., with Ballif, 358 (Pel.)
Ghetti, G., (668) (R.F.)
Ghose, G., 462 (Fev.)
Ghosh, G., 354 (Bb.)
Ghosh, N., 743 (K.A.)
Ghosh, N. N., with Chopra & Gupta, 566 (Misc.)
Ghosh, S., 569, (600) (Misc.)
——, Chopra, R. N. & Chatterjee, N. R., 569 (Misc.)
——, with —— & Dutt, 566 (Misc.)
——, with Henderson & De, 617 (Lep.)
——, with Megaw & Chatterjee, 23 (Mal.)
Ghosh, U., 967 (Bb.)
Giagni, with Delamare, 866 (G.V.)
Giauni, G., 948 (Mal.)
Gibb, D. F., 277 (Z.)
Gibbs, O. S., 569 (Misc.)
Gibson, A. G., with Fawcett, 768 (Myc.)
Giglioli, G., 864 (G.V.)
Gilbert, R. & Coleman, M. B., 439 (Und.) 895 (Dys.)
Gildemeister, E. & Karmann, P., 219 (Rab.)
Gill, D. G., Kiker, C. C. & Sims, L. C., 919 (Mal.)
Gill, J. M., with Kerrin, 479 (Dys.)
Gillet, H., 409 (Misc.)
Gilmer, W. P., with Wilson, 1041 (Lep.)
Gilroy, J. C., 146 (Am.)
Gimlette, J. D., 168 (B.R.)
Ginés, with Pittaluga, Martinez Cepa & Torrijos, 917 (Mal.)
Ginestet, G., 969 (Hel.)
Giordano, A. S., with Sensenich, 60 (Und.)
Giordano, M., 237 (Z.), 666 (R.F.), 684 (Der.), 1040 (Lep.)
Girard, G., 636 (Pl.)
—— & Robic, J., 861 (Y. & S.)
Giraud, A., with Foley & Catanei, 363 (Mal.)
Giraud, P. & Massot, M., 316 (K.A.)
Giraudeau, R., with Jeanselme & Bureau, 614 (Lep.)
Giroud, P., with Blanc & Caminopetros, 839 (Fev.)
Githens, T. S. & Butz, L. W., 779 (Z.)
Giugni, F. & Savorini, G., 57 (Und.)

Gladin, S., 265 (Z.)
Gloster, T. H., 735 (Rab.)
Glover, W. E., 268 (Z.)
Gnedina, M. P. & Statirova, N. A., (558) (Hel.)
Goiffon, R., 571 (Misc.)
Goldberger, J., 961 (Pel.)
——, Wheeler, G. A., Sydenstricker, E., King, W. I. & others, 356, 963 (Pel.)
Gold Coast, Ann. Rep. Med. Res. Inst. 389 (Lab.)
——, Rep. on Med. & San. Dept. 646 (Y.F.)
Goldie, H., 840 (Fev.), 971 (Hel.)
Gollini, A., (600) (Misc.)
Gomes, J. M. & Junior, J. D. de P., 341 (Lep.)
—— & do Pateo, Jr., J. D., 617 (Lep.)
—— & Pessoa, S. B., 685 (Der.)
——, with Silveira, 1041 *bis* (Lep.)
Gontscharow, G. D., 815 (Z.)
González Azcune, F., with Alonso Alonso, (949) (Mal.)
Gonzalez Medina, R., with Covisa, 315 (K.A.)
Goré, S. N., with Mackie, 501 (Sp.)
——, with —— & Wadia, 497 (Sp.)
Gori, P., with Manieri, 663 (R.F.)
Gosio, R., 1029 (Bl.)
Goyle, A. N., 98 (Pl.)
Grady, A. G., 257 (Z.)
Graf, 703 (S.S.)
Graham, J. D., 83 *bis*, 89, 90 (Chl.), 329 (K.A.), (642) (Pl.)
Grant, H. G., 919 (Mal.)
Grassmück, E., 891 (Dys.)
Gray, G. M., 1015 (Bl.)
Gray, J. D. A., 116 (R.F.)
Greco, N. V., 343 (Lep.)
Greeley, H. P., with Stovall, 761 (Myc.)
Green, F. W., with Jackson, van Hovenberg, Filby & Clarke, 37 (Mal.)
Green, R., 333, 614 (Lep.), 380, 931 *bis*, (949) (Mal.), 759 (Myc.)
Greenway, D., 152 (Am.), 232 (Z.)
——, with Castex, 232, (258) (Z.)
——, with ——, Greenway, D. F. & Urdaniz, 476 (Am.)
Gregg, A. L., 606 (B.R.)
Grekow, A. D., (642) (Pl.)
Greppi, L., 895 (Dys.)
Grey, C. G., 1025 (Bl.)
Gridnew, V. M., 45 (Fev.)
Griffitts, T. H. D., 919 (Mal.)
Gromaschewskij, L. W. & Schuchat, J. A., 268 (Z.)
Gross, M., 598 (Misc.)
Gudger, E. W. & Breder, Jr., C. M., 791 (Z.)
Guelmino, D., 917 (Mal.)
Guénolé, A., with Tournier, 455 (Fev.)
Guerrant, N. B., with Salmon & Hays, 359 (Pel.)
Guija Morales, E., (38) (Mal.)
Guitarte, A., (38) (Mal.)
Guldberg, G., 183 (G.V.)
Gupta, A., 677 (Der.)
Gupta, A. K. D., with Knowles, Gupta, B. M. D. & Gupta, U., 148 (Am.)
Gupta, B. M. D. & Dikshit, B. B., 752 (K.A.)
——, with Knowles, 139 (Fev.), 798 (Z.)
—— with ——, Gupta, A. K. D. & Gupta, U., 148 (Am.)
Gupta, J. C., with Chopra & Choudhury, 571 (Misc.)
——, with —— & Ghosh, 566 (Misc.)
——, with —— & Mullick, 171 (Y. & S.)
——, with —— & Pillai, 564 (Misc.)
Gupta, U., with Knowles, Gupta, A. K. D., & Gupta, B. M. D., 148 (Am.)
Gushue-Taylor, G., 608 (Lep.)
Gutiérrez Lara, J., 211 (S.S.), (558) (Hel.)
Gutzewitsch, A. W., with Magnitsky, 816 (Z.)
Gwéléssiany, J., with Alexeieff, 797 (Z.)

## H

Habermann, R. & Serefis, S., (388) (Mal.)
Habs, H., 61 (Und.)
Hackett, L. W., 919 (Mal.)
——, with Missiroli, (824) (Z.)
Haddon, E. C., with Duke & Hall, 262 (Z.)
Hadenfeldt, A., with Seelemann, 443 (Und.)
Hadjigeorges, A., with Papadopoulo & Yoel, 835 (Fev.)
Hahn, M. & Hirsch, J., 628 (Chl.)
Hakki, I., 527 (Hel.)
Hall, G. N., with Duke & Haddon, 262 (Z.)
Hall, R. N., 174 (Y. & S.)
Hamilton, R. G., 919 (Mal.)
Hamilton-Browne, E., 752 (K.A.)
Hamlyn-Harris, R., 808, 809 (Z.)
Hanafin, J. B., 581 (Misc.)
Hance, J. B., 368 (Mal.)
—— & Pershad, J., 580 (Misc.)
Hanschell, H. M., 703 (S.S.), 865 (G.V.)
Hanson, H., 11 (Mal.), 994 (Y.F.)
Hara, Y., 778 (Z.)
Harada, N., with Mikawa & Nomura, (950) (Mal.)
Harazoff, T. G., (38) (Mal.)
Hardy, A. V., 439, 827 (Und.)
Harley-Mason, R. J., 595 (Misc.)
Harris, Jr., H. E., 973 (Hel.)
Harris, N. G., 386 (Mal.)
Harrison, W. T., with Jackson, 273 (Z.)
Hartoch, O. & Rothermundt, M., 662 (R.F.)
Harvey, F. M., with Newham, 561 (Misc.)
Haschnow, G., with Tanew, 933 (Mal.)
Hase, A., 277, 822 (Z.)
Hasegawa, K., 981 (Hel.)
Haslé, G., 623 (Lep.)
Hassall, A., with Stiles, 241 (Z.)
Hasselmann, C. M. & Hasselmann-Kahlert, M., 371 (Mal.)
Hasselmann-Kahlert, M., with Hasselmann, 371 (Mal.)
Hauduroy, P. & Ghalib, A., 638 (Pl.)
Hauser, G. H., 223 (Rab.)
Hay, W. D., 226 (Rab.)
Hayashi, F., 341 (Lep.)
Hays, I. M., with Salmon & Guerrant, 359 (Pel.)
Heagerty, J. J., 345 (B.R.)
Heatly-Spencer, J., 47 (Fev.)
Hecht, O., 247 (Z.)
Hecht-Eleda, M., 384 (Mal.)
Hegh, E., 601 (B.R.)
Hegler, C., 494 (Sp.)
Hegner, R., 229, 802 (Z.)
——, Shaw, Jr., E. H. & Manwell, R. D., 24 (Mal.)

Heideman, M. L., with Cole, 874 (Am.)
Hein, G. E. & Merrill, J., 960 (Pel.)
Heinemann, H., 854 (Y. & S.)
Helm, R., 717 (S.S.)
Helmy, M. M., 971 (Hel.)
——, with Augustine, Nazmi & McGavran, 553 (Hel.)
Hemphill, R., 635 (Pl.)
Henderson, J. M., 1036 (Lep.)
—— & Chatterji, S. P., 339 (Lep.)
——, De, N. K. & Ghosh, S., 617 (Lep.)
——, with Muir, 1036 (Lep.)
——, with Napier, 1036 (Lep.)
Hennessy, P. H., 510 (Oph.)
Henry, A. F. X., 383 *ter*, 940 *ter* (Mal.)
Henry, E., with van den Branden, (951) (Mal.)
d'Hérelle, F., 86 (Chl.)
Hérivaux, 190 (S.S.)
Hermans, E. H., 598 (Misc.), 857 (Y. & S.)
Hermans, E., with de Langen, 1031 (Lep.)
Herms, W. B., 243 (Z.)
Herrick, C. A. & Emery, F. E., 549 (Hel.)
Herrmann, G. & Hlisnikowski, H., 386 (Mal.)
Herrmann, O., 225 *bis*, 727 (Rab.)
—— & Malinina, A., 9 (Mal.)
Hershfield, A. S., Kibler, O. A., Colby, S., Koenig, M. T., Schmid, O. W. & Saunders, A. M., 675 (Fev.)
Hertig, M., with Young, 741 (K.A.)
Herzenberg, L., 725 (Rab.)
Hesse, E., 734 (Rab.)
Hewetson, W. M., 1018 (Bl.)
Heydon, G. M., 554, 989 (Hel.)
Hicks, E. P., 264 (Z.)
Hildebrand, P., 281 (Hist.)
Hill, L. R., with Jaffé, 766 (Myc.)
Hill, R. B., 921 (Mal.)
—— & Benarroch, E., 371 (Mal.)
Hill, R. C., 690 (Der.)
Hillel-Yofé, 1017 (Bl.)
Hilleret, (903) (Dys.)
Hillsman, J. A., Wilshusen, H. F. & Zimmerman, H. M., 184 (C.Bu.)
Hindle, E., 292 (Y.F.), 311 (K.A.)
——, assisted by Chow, F. L., 910 (Mal.)
Hinman, E. H., with Matheson, 813 (Z.)
Hirasawa, I., 531 (Hel.)
Hirsch, J., 628 (Chl.)
——, with Hahn, 628 (Chl.)
Hirst, A. S., 822 (Z.)
Hizon, J., with Dionisio Gutierrez, 111 (Der.)
Hlisnikowski, H., with Herrmann, 386 (Mal.)
Hoare, C. A., 212 (S.S.)
Hockett, A. J., 929 (Mal.)
Hodges, R. H., 469 (Am.)
Hodgson, E. C. & Sen, R. T., 322 (K.A.)
——, —— & Das, C., 321 (K.A.)
Hoeltzer, R. R., with Aristowsky, 660 (R.F.)
Hoeppli, R., 1019 (Bl.)
Hoffman, F. L., 919 (Mal.)
Hoffman, W. A., Marín, R. A. & Burke, A. M. B., 544 (Hel.)
——, with Taliaferro & Cook, 534, 535 (Hel.)
Hoffman, W. H., 542 (Hel.)
Hoffmann, C. C., 700 (S.S.)
Hoffmann, W. H., 104 (Der.), (309) *bis* (Y.F.), 619 (Lep.), 764 (Myc.), 952 (B.R.), 998, 1008 (Y.F.)
—— & Embil, V. A., 791 (Z.)
—— & Jahnel, F., 308 (Y.F.)
Hoffmann, W. H. & Ramos Baez, P., (1044) (Lep.)
Hoflin, J. W., 36 (Mal.)
Hogue, M. J., 235 (Z.)
Hohenadel, B., with Kuczynski, 296 (Y.F.)
Holmes, M. J., 37 (Mal.)
Honig, P. J. J. & Swellengrebel, N. H., 282 (Hist.)
Hopf, G., 10 (Mal.)
Hopkins, R., 1036 (Lep.)
—— & Denney, O. E., 607 (Lep.)
Horn, L., 943 (Mal.)
Hornby, H. E., 711 (S.S.)
Hornus, P., 652 (R.F.)
——, with Nicolle & Anderson, 657 (R.F.)
Horovitz, A. & Sautet, J., 936 (Mal.)
Horowitz, with Jeanselme, (344) (Lep.), 600 (Misc.)
Hoshi, N., 478 (Dys.)
Hoshizaki, S., 162 (Dys.)
Hosoya, S., with Stéfanopoulo, 129 (R.F.), 483 *bis* (Dys.)
Hotta, T., 221 (Rab.)
Hotta, Y., 30 (Mal.)
Hötte, F. A., 415 (Misc.)
van Hovenberg, H. W., with Jackson, Filby, Green & Clarke, 37 (Mal.)
Howard, H. H., 552 (Hel.)
Howden, A. L., 477 (Dys.)
Howell, A. T., (600) (Misc.)
Hu, C. H., Huie, D. & Lee, C. U., 742 (K.A.)
—— & Lee, C. U., 742 (K.A.)
——, Reimann, H. A. & Kurotchkin, T. G., 765 (Myc.)
Huddleson, I. F. & Abell, E., 73 (Und.)
Hudson, E. H., 177 (Y. & S.)
Hudson, N. P., with Bauer, 303 (Y.F.)
——, Bauer, J. H. & Philip, C. B., 647 (Y.F.)
——, Philip, C. B. & Davis, G. E., 1005 (Y.F.)
Huertas, F., (359) (Pel.)
Huet, L., with Jeanselme & Lotte, 106 (Der.)
Huie, D., with Hu & Lee, 742 (K.A.)
Huinink, A. S. B., with Schuurman, 912 (Mal.)
Huisman, R. G. J. P., 814 (Z.)
Huizenga, L. S., 1034 (Lep.)
Hulshoff, A. A., 373 (Mal.)
——, with Olivier, 20 (Mal.)
Hunt, R., McCann, W. S., Rowntree, L. G., Voegtlin, C. & Eggleston, C., 378 (Mal.)

## I

Iagnov, S., 595 (Misc.)
Ichok, G., 457 (Fev.)
von Ihering, R. (258) (Z.)
Iio, J., (489) (Dys.)
Imahasi, T., 792 (Z.)
Imai, B., 535 (Hel.)
——, with Miyaji, 535 (Hel.)
Indian Medical Gazette, 106 (Der.), 313 (K.A.)
Infurna, G., 581 (Misc.)
Ingram, A. & de Meillon, B., 809 (Z.)
Inniss, K. U. A., 276 (Z.)
Ionesco-Mihaiesti, C., Combiesco, D., Condrea, P. & Calalb, G., 156 (Dys.)
Iriarte, D. R., 227, 257 (Z.)
—— & Salas, L. M., 864 (G.V.)
Irvine, G. M., (949) (Mal.)
Isaacs, R., Sturgis, C. C. & Smith, M., 540 (Hel.)

Ishida, I., 235 (Z.)
Ishizu, Y., 137 (Fev.)
Ismail, A., (600) (Misc.)
Ito, K., with Matsumura et al, 965 (Bb.)
Iwanowa, D. A., 116 (R.F.)
Iyengar, M. O. T., 3 (Mal.), 244, 250, 265, 815 (Z.)
—— & Sur, P., 3 (Mal.)
Izar, G., 64 (Und.)
Izquierdo Salazar, A., (165) (Dys.)

## J

Jack, R. W., 260 (Z.)
Jackson, D. & Harrison, W. T., 273 (Z.)
Jackson, L. E., van Hovenberg, H. W., Filby, E. L., Green, F. W. & Clarke, J. L., 37 (Mal.)
Jacobson, H. P., 765 (Myc.)
Jacoby, C., 901 (Dys.)
Jacocks, W. P., 552 *bis* (Hel.)
——, with Carter, 947 (Mal.)
Jaffé, R. H. & Hill, L. R., 766 (Myc.)
Jaguboff, F., 973 (Hel.)
Jahnel, F., with Hoffmann, 308 (Y.F.)
—— & Lange, J., 175 (Y. & S.)
Jakimow, W. P., 125, 661 (R.F.)
Jakob, Fialho, A., Villela, A. & E. L., 298 (Y.F.)
James, W. M., 140 *bis* (Am.), 575 (Misc.)
Jancsó, N., 388 (Mal.)
Japan Medical World, (642)( Pl.)
Jausion, H., 126 (R.F.)
Jean, L., 11 (Mal.)
—— & van Nitsen, R., 11 (Mal.)
Jeanselme, Burnier & Eliascheff, 856 (Y. & S.)
—— & Horowitz, (344) (Lep.), (600) (Misc.)
——, Huet, L. & Lotte, 106 (Der.)
Jeanselme, E., 624 (Lep.)
——, Giraudeau, R. & Bureau, Y., 614 (Lep.)
Jemma, R., 740 (K.A.)
Jensen, J. P., (74) (Und.)
Jensen, K. A., with Madsen, 890 (Dys.)
Jensen, V., (949) (Mal.)
Joannidès, G., with Blanc & Caminopetros, 761 (Myc.)
Joannidès, G. S., 837 (Fev.)
Joenoes, M. I., with Radsma, 578 (Misc.)
Johns, F. M., 680 (Der.)
Johnson, H. A., with LePrince, 919 (Mal.)
Johnson, T. L., 719, 720 (S.S.)
Johnson, W. B. & Lester, H. M. O., 701 (S.S.)
—— & Lloyd, L., 259 (Z.)
Johnsson, J. W. S., 285 (Hist.)
Jones, G., with Duhig, 278, 279 (Z.)
Jonesco, D. & Teodosio, V., 728 (Rab.)
—— & Valter, V., 729 (Rab.)
Jordan, D. S. (258) (Z.)
Jorge, R., 95 (Pl.)
Joseph, A., with Buschke, 104 (Der.)
Jourdran, E. & Le Meillour, 964 (Bb.)
Journal of Oriental Medicine, (642) (Pl.)
Joyeux, C. & Baer, J. G., 975, 976 (Hel.)
Jungmann, P., 300 (Y.F.)
Junior, J. D. de P., with Gomes, 341 (Lep.)
Justesen, P. T., 797 (Z.)

## K

Kabelík, J., 387 (Mal.)
Kadaner, M., 37 (Mal.), 174 (Y. & S.), 646 (Y.F.)
Kaganova, S. S. with Kritschewski, 711 (S.S.)
Kakinuma, G., with Matsumura et al, 965 (Bb.)
Kalmikoff, E. S., (949) (Mal.)
Kalthofen, A., 863 (G.V.)
Kamal, H., 39 (Fev.)
Kamath, A. V., 355 (Bb.)
Kampmeier, R. H., 57 (Und.)
Kanagarayer, K., 550 (Hel.)
Kanao, R., with Matsumura et al, 965 (Bb.)
Kandelaki, S., (949) (Mal.)
——, Okropiridse, B. & Abuladse, S., (949) (Mal.)
Kaneko, R., 307 (Y.F.)
Kangragsat, S., 275 (Z.)
Kaplan, B., with Williamson & Geiger, 870 (Am.)
Karamchandani, P. V., 19 (Mal.)
Karger, P., 975 (Hel.)
Kariboff, N., (993) (Hel.)
Karibow, N., 818 (Z.), 973 (Hel.)
Karmann, P., 737 (Rab.)
——, with Gildemeister, 219 (Rab.)
Karve, D. S., 924 (Mal.)
Kasauli: Pasteur Institute of India, 223, 735 (Rab.)
Kathe, 132, 672 (R.F.)
Kauders, O., 30 (Mal.)
Kauntze, W. H., 395 (Lab.)
Kavanaugh, C. N., 826 (Und.)
Kawashima, K., with Matsumura et al, 965 (Bb.)
Keigeluchis, I., 747 (K.A.)
Keil, E. & Unna, Jr., P., 620 (Lep.)
Keining, E. & Serefis, S., 385 (Mal.)
Kellaway, C. H., 774 *ter*, 780 (Z.), 978 (Hel.)
Kellogg, C. R., with Faust, 786 (Z.)
Kenya, 395 (Lab.)
Kern, R. A., 60 (Und.)
Kerr, I., 1039 (Lep.)
Kerrin, J. C., 153 (Dys.)
—— & Gill, J. M., 479 (Dys.)
Kessel, J. F., 151 (Am.), 229, 230, 235 (Z.)
Keyworth, W. D., 367 (Mal.)
Khalid, Z., 161 (Dys.)
Khalil, M., Nazmi, M., Peter, F. M., El Din, M. S. & El Betash, M. H., 972 (Hel.)
Khodukin, N. I., 324 (K.A.)
Khouri, J., 486 (Dys.), 871, 881 (Am.)
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——, with Regendanz, 795 (Z.)
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King, H. H. & others, 909 (Mal.)
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Kingsley-Ward, S., 909 (Mal.)
Kipschidse, N., 488 (Dys.)
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——, Dove, W. E. & White, G. F., (993) (Hel.)

Kirillova-Oudintsova, A. W., (558) (Hel.)
Kirk, J. B., 543 (Hel.)
Kirkpatrick, H., 504 (Oph.)
Kirwan, E. W. O'G., 370 (Mal.), 506 (Oph.)
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Kobayashi, T., 555 (Hel.)
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Koningin Wilhelmina Jubileum Stichting, 415 (Misc.)
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Kopke, A., 189 (S.S.)
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Laidlaw, P. P., Dobell, C. & Bishop, A., 469 (Am.)
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Lane, C., 605 (B.R.), 981 (Hel.)
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Lange, J., with Jahnel, 175 (Y. & S.)
de Langen, C. D., 163 (Dys.), 543 (Hel), 616 *bis* (Lep.), 988 (Hel.)
—— & Hermans, E., 1031 (Lep.)
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Legendre, J., 2 *bis* (Mal.), 650 (Y.F.), 807 (Z.)
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Leisermann, L. I., 377 (Mal.)
Leitch, J. N., 957 (B.R.)
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Le Meillour, with Jourdran, 964 (Bb.)
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Leroy, D., with Voizard, 107 (Der.)
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Li, C. & Faust, E. C., 539 (Hel.)
Lian, S. B., 504 (Oph.)
Lichtenstein, A., 373 (Mal.), 1018 (Bl.)
——, with de Langen, 430 (B.R.)
Lie, H. P., 611 (Lep.)
Leibman, E., with Kligler, 364 (Mal.)
Liégeois, R., with Le Bourdellès, 383, 941 (Mal.)
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Lindtrop, H., 56 (Und.)
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Lins, S. A., 995 (Y.F.)
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Lipowska, L., with Przesmycki & Sierakowski, 157 (Dys.)
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Lissner, L., 856 (Y. & S.)
Lissovsky, L. L., with Schourenkowa, 232 (Z.)
Lister, F. S., 393 (Lab.)
Liu, H. L., 536 (Hel.)
Liu, K.-B., 150 (Am.)
Livierato, S. & Vagliano, M., 452 (Fev.)
Ljachowetzky, A. M., 377 (Mal.)
Lloyd, L., with Johnson, 259 (Z.)
——, with Lester, 263 (Z.)
Lloyd, R. B., 593 (Misc.)
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Lloyd, W. D. M., with Sawyer & Kitchen, 1007 (Y.F.)
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Löffler, W., 826 (Und.)
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Lokhov, M., with Semikoz, 641 (Pl.)
Lommel, J., 479 (Dys.)
Long Island Medical Journal, 219 (Rab.)
Lopez Rizal, L., 349 (Bb.)
Lorando, N., 666 (R.F.)
Loreti, F., 372 (Mal.)
Losaberidse, E., 970 (Hel.)
Lotte, with Jeanselme & Huet, 106 (Der.)
Low, G. C., 490 (Sp.)
——, Cooke, W. E. & Martin, P. H., 1023 (Bl.)
Lowe, J., 613, 1037 (Lep.)
Lowenberg, K., 220 (Rab.)
Lozano, P. & Peña, M., 651 (R.F.)
de Luca, B., 21 (Mal.)
Luengo Garcia, F., 1035 (Lep.)
di Lulio, O., 686 (Der.)
di Lullo, O., 109 (Der.)
Lumbau, D., with Fermi, 729 (Rab.)
Lumbroso, U., with Sparrow & Lapidari, 665 (R.F.)
Luna Gañán, J., 12 (Mal.)
Lunevski, with Ballif, 381 (Mal.)

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Lusena, M., 768 (Myc.)
Lwoff, M., 236 (Z.)
Lynch, K. M., 232 (Z.)

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—— & Slanetz, C. A., 74 (Und.)
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McCarrison, R., 596 (Misc.)
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McCulloch, W. E., 109 (Der.)
McCutcheon, O., 909 (Mal.), 1017 (Bl.)
McDaniel, J. C., 404 (Lab.)
McDermott, E. N., 136 (Fev.)
McGavran, E. G., with Augustine, Nazmi & Helmy, 553 (Hel.)
McGrath, M. J., 476 (Dys.)
McGuire, C., with Acton, 682 (Der.)
Macht, D. I., 619 (Lep.)
M'Hutchison, G. B. & Duff, W. R., 18 (Mal.)
Machwiladse, N., 357, 962 (Pel.), 387 (Mal.)
McIntosh, J. A., 181 (G. V.)
McKendrick, A. G., 215, 725 (Rab.)
Mackenzie, I., 9 (Mal.)
Mackenzie, J., 868 (B. R.)
Mackenzie, R., 547 (Hel.)
Mackie, F. P., 398 (Lab.), 501 (Sp.)
—— & Chitre, G. D., 498, 499 (Sp.)
—— & Fairley, N. H., 500 (Sp.)
——, with —— & Billimoria, 491 (Sp.)
—— & Goré, S. N., 501 (Sp.)
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McKinley, E. B., 673 (R.F.)
Mackinnon, J. E., with Talice, 761 (Myc.)
McKnight, R. B., 469 (Am.)
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Madsen, T., 58 (Und.)
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de Magalhães, O., 276 (Z.), 758 (Myc.)
Magath, T. B., 539 (Hel.)
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Magid, M. I., 387 (Mal.)
Magnitsky, W. J. & Gutzewitsch, A. W., 816 (Z.)
Mahler, P., 488 (Dys.)
Majid, A., with Young, (258) (Z.), (951) (Mal.)
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Makaroft, A. K., 820 (Z.)
Malinina, A., with Herrmann, 9 (Mal.)
Malone, R. H., 735 (Rab.)
Manai, A., 594 (Misc.)
Manalang, C., 367 (Mal.)
Mandoul, 813 (Z.)
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Manson-Bahr, P., 159 (Dys.), 375, 945 (Mal.), 429 (B.R.), 874 (Am.), 981 (Hel.)
—— & Kilner, T. P., 879 (Am.)
—— & Willoughby, H. M., 145, 873 (Am.), 825 (Und.)
Manuwa, S. L. A., 268 (Z.)
Manwell, R. D., 801 (Z.)
——, with Hegner & Shaw, Jr., 24 (Mal.)
Maplestone, P. A., 545, 548, 553 (Hel.)
Marcandier, with Plazy & Marcon, 457 (Fev.)
Marcel, with Sartory & Mayer, 683 (Der.)
Marchal, G., with Flandin & Langlois, (949) (Mal.)
Marchiafava, E., 80 (B.R.)
Marchoux, E., 1005 (Y.F.), 1036 (Lep.)
Marçon, with Plazy, 457 (Fev.)
——, with —— & Marcandier, 457 (Fev.)
Maréchal, H., (950) (Mal.)
Marginesu, P., 737 (Rab.), 944 (Mal.)
Maria Macera, J. & Carreno, C., (993) (Hel.)
Marie, A. C., 729 (Rab.)
—— & Mutermilch, S., 217 (Rab.)
Marin, R. A., with Costa-Mandry, 466 (Am.)
——, with Hoffman & Burke, 544 (Hel.)
Marino, V., 633 (Pl.)
Markianos, J., 617, 621 *bis* (Lep.)
Markoff, W., 114 (R.F.)
Markowski, S. & Legezynski, S., 221, 225, 735, 737 (Rab.)
Marneffe, H., 340, (344), 621 (Lep.)
Marquand, G., 573 (Misc.)
Marras, A. & Frau, A., 1042 (Lep.)
Marras, F. M., 631 (Chl.)
Martelli, B. N. & Mascagni, G., 25 (Mal.)
Martens, A. H. A. & Koers, C. H., 235 (Z.)
Martin, E., with Roch & Monedjikova, 63 (Und.)
Martin, H., 175 (Y. & S.)
Martin, P. H., with Low & Cooke, 1023 (Bl.)
Martindale, W. H., 470 (Am.)
Martinez Cepa, with Pittaluga, Torrijos & Ginés, 917 (Mal.)
Martini, E., 35 (Mal.), 81 (B.R.), 588 (Misc.), (824) (Z.)
Maruaschwili, M., 365 (Mal.)
Marzinowsky, E. I., 325 (K.A.)
——, Pickoul, J. N. & Balaschewa, M. T., 373 (Mal.)
Masataka, M. & Teiryo, T., (344 *quat*) (Lep.)
Mascagni, G., with Martelli, 25 (Mal.)
Massa, M., 382 (Mal.)
Massias, C., 220 (Rab.)
Massot, M., with Giraud, 316 (K.A.)
Matheson, R. & Hinman, E. H., 813 (Z.)
Mathewossian, S. T., 227 (Z.)
Mathis, C., 116 *bis* (R.F.)
——, with Nicolle & Anderson, (128) (R.F.)
——, with Sellards, 647 (Y.F.)
Mathis, M., 814 (Z.)
Matjuschenko, B., (38) (Mal.)
Matsumoto, K., Ando, K. & Shiraiwa, T., 87 (Chl.)
—— & Seki, T., 481 (Dys.)

Matsumura, S., 965 (Bb.)
——, Kakinuma, G., Kawashima, K., Tanikawa, K., Ochiai, S., Miyata, R., Fujisaki, K., Kanao, R., Noguchi, K., Aoki, K., Sato, T., Ito, K. & Suzuki, M., 965 (Bb.)
Matsuoka, F., (600) (Misc.)
da Matta, A., 610, (625) *bis* (1044) *bis* (Lep.)
Mattos, A., 163 (Dys.)
Maurer, S., with Richter & Eyl, 540 (Hel.)
Maurice, G. K., 700 (S.S.)
Maxcy, K. F., 378 (Mal.)
Maxwell, J. L., 332, 339 (Lep.), 511 (B.R.), 576 (Misc.)
Mayeda, S., 568 (Misc.)
Mayer, M., 196 (S.S.), (523), (524) (Z.), 753 (K.A.), 783 (B.R.)
——, with Sartory & Marcel, 683 (Der.)
Mayne, B., (38 *bis*) (Mal.), 253 (Z.)
Mayr, J. K., 385 (Mal.)
Mazza, S., 22 (Mal.)
—— & Rickard, E. R., 366 (Mal.)
—— & Trelles, R., (38) (Mal.)
Mazzeo, M., 484 (Dys.)
Mebius, J., 964 (Bb.)
Medeiros, M., 102 (Der.)
Medical Journal of Australia, 773 (Z.)
Medical & Scientific Archives of the Adelaide Hospital, 461 (Fev.), 776 (Z.)
Medsmariaschwili, I., (950) (Mal.)
Megaw, J. W. D., Bhattacharji, S. P. & Paul, B. K., 353 (Bb.)
——, Ghosh, S. & Chatterjee, N. R., 23 (Mal.)
—— & Rao, S. S., 49 (Fev.)
Meihuizen, F. H., (388) (Mal.), 634 (Pl.)
Meillon, B., with Ingram, 809 (Z.)
Meleney, H. E., 117, 661 (R.F.)
——, Bishop, E. L. & Roberts, F. L., 919 (Mal.)
Melkersson, E., (676) (Fev.)
Mello, A. da S., (309) *ter* (Y.F.)
de Mello, E. J., 995 (Y.F.), 1032 (Lep.)
de Mello, F., 149 (Am.), 933 (Mal.)
—— & da Cruz, L. J. C., 475, 878 (Am.)
—— & Fialho, A., 123 (R.F.)
—— & Pachêco de Figueiredo, F., (489) (Dys.)
—— & Vernencar, H. P., 25 (Mal.)
Melnotte, P., (165) (Dys.)
——, with Deschiens, 465 (Am.)
——, with Lacaze, 468 (Am.)
Mendelson, R. W., 102 (Der.)
——, with Castellani, 866 (G.V.)
Mendes, N. O., with de Assis, 156 (Dys.)
Mendoza-Guazon, M. P., 152 (Dys.)
Menk, W., 16 (Mal.), 404 (Lab.), 1013 (Bl.)
Mense, C., 867 (B.R.)
Menten, M. L. & Krugh, H. M., (489) (Dys.)
Menton, J., 155, 890 (Dys.)
Merighi, G., with Schiassi, 19 (Mal.)
Merrill, J., with Hein, 960 (Pel.)
Mertens, W. K., 583 (Misc.)
Meslay-Coirre & Cochez, 330 (K.A.)
Mesnard, J., 327 (K.A.), 589 (Misc.)
Metcalf, M. M., 785 (Z.)
Metelkin, A., 524 (Z.)
Mételkine, A., 609 (Lep.)
Metzger, H., (489) (Dys.)
Mexico, 425 (B.R.)
Meyer, H., 802 (Z.)
Mglej, S., with Finik, 736 (Rab.)
Michael, J. C., (690) (Der.)
Michailowa, M. A. & Oudintzew, G. N., 240 (Z.)
Michel, P., with Gaté, 862 (C.Bu.)
Michelson, E., 800 (Z.)
Mickle, F. L., with McAlpine, 70 (Und.)
Miessner, H. & Baars, G., 225 (Rab.)
Migneco, G., with Sanfilippo, 445 (Und.)
Miguens, J., 493 (Sp.)
Mikawa, T., Nomura, G. & Harada, N., (950) (Mal.)
Millan Muñoz, J., (388) (Mal.)
Miller, Jr., H. M. & Dawley, C. W., 540 (Hel.)
Miller, J. E., 841 (Fev.)
Miller, M. W., 150 (Am.)
Milne, C. J., 581 (Misc.)
Minamizaki, T., 556 (Hel.)
Minina, R. P., (330) (K.A.)
Missiroli, A., 34 (Mal.)
—— & Hackett, L. W., (824) (Z.)
—— & Siniscalchi, R., (950) (Mal.)
Missra, J. N., (1044) (Lep.)
Misuraca, S., 56 (Und.)
Mitard, L., with Boulay & Lhuerre, 24 (Ma.)
Mitchell, J., 964 (Bb.)
Mitra, A. C., 572 (Misc.)
Mitrofanova, J., (824) (Z.)
Mitrophanova, J., with Beklemischev, 251 (Z.)
Mitsuhashi, C., 163 (Dys.)
Mittra, P., with Cochrane, 340 (Lep.)
Miyaji, S. & Imai, B., 535 (Hel.)
Miyata, R., with Matsumura et al, 965 (Bb.)
Mochtar, A., 132 (R.F.)
Moghilevskaia, B., with Zlatogoroff, 99 (Pl.)
Moghilewskaja, B. I., with Zlatogoroff, 640 (Pl.)
Mogilevskaya, B., with Zlatogorov, 640 (Pl.)
Molodtzova, P., 642 (Pl.)
Monacelli, M., 336 (Lep.)
Moncarey, 534 (Hel.)
Moncorvo, F., 298 (Y.F.)
Mondain, A., with Legendre, 379 (Mal.)
——, with —— & Razafindramamba, 949 (Mal.)
Mondini, E. M., (388) (Mal.)
Monedjikova, V., with Roch & Martin, 63 (Und.)
Monje, C. & Weiss, P., 55 (Fev.)
Monteiro, J. L., 998 (Y.F.)
Montel, L. R., 851 (Y. & S.)
Montel, M. L. R., 472 (Am.)
—— & Vielle, A., 147 (Am.)
Montes Pareja, J., 882 (Am.)
Montgomery, H., (344) (Lep.)
Montpellier, J., Catanei, A. & Cattoir, 769 (Myc.)
Mooser, H., 50 (Fev.), 738 (Rab.)
Mora, A. D., 408 (Misc.)
Morales Villazón, N., with Uriarte, 635 (Pl.)
Moralis, P. A., 1030 (Bl.)
Morel, C. & Tapie, J., 873 (Am.)
Morin, H. G. S., (38) (Mal.)
—— & Pirot, R., 455 (Fev.)
Morishita, K., 718 (S. S.)
Morison, J., 222 (Rab.)
——, with Choudhury, 901 (Dys.)
Moritsch, P. & Pirker, H., 274 (Z.)
Morrison, H., 391 (Lab.)
Morrison, N., 275 (Z.)
Morton, H. M., 507 (Oph.)
Moses, A., 597 (Misc.), 1004 (Y.F.)

Moskwin, J. A., 823 (Z.)
Motta, C., 328 (K.A.)
Motta, J., 613 (Lep.)
Mouchet, R., 293 (Y.F.)
Moufel, P. P., (824) (Z.)
Mounier, with Garin & Doubrow, 991 (Hel.)
Moutoussis, K., 837 (Fev.)
Mtschedlidse, W., (950) (Mal.)
Muffel, P. P., 377, 918 (Mal.)
Muggia, A., 895 (Dys.)
Mühlens, P., 583 (Misc.), 702 (S.S.), 900 (Dys.)
——, with Delbanco & Vogel, 984 (Hel.)
Muir, E., 612 (Lep.)
—— & Henderson, J. M., 1036 (Lep.)
Muller, F. W., with Webb, 728 (Rab.)
Müller, M., 281 (Hist.)
Mullick, M. N., with Chopra & Gupta, 171 (Y. & S.)
——, with Napier, 321 (K.A.)
Mulligan, H. W., 936 (Mal.)
Muniz, J., with da Cunha, 55, 463 (Fev.), (258) (Z.), 295, 303, 308, 646 (Y.F.), (902) (Dys.)
Murakami, K., 887 (Dys.)
Murayama, I., 665 (R.F.)
Murgatroyd, F., with Owen, 1016 (Bl.)
Murphey, E. E., 919 (Mal.)
Murphy, R. A., with Strickland, Chowdhury, Dodds-Price, Forsyth, Smith & Williams, 4 (Mal.)
Mutermilch, S., with Marie, 217 (Rab.)
Mya, T., 547 (Hel.)
Myers, J. G., 816 (Z.)

## N

Nag, S. C., 1026 (Bl.)
Naegeli, T., 782 (B.R.)
Naeslund, C. & Ströman, R., 91 (Pl.)
Nagai, S., with Ogata & Unno, 53 (Fev.)
Nagata, S., with Tsuchiya, 487 (Dys.)
Nagayo, M., 527 (Hel.)
Najera, L., 785 (Z.)
Nakamura, H., with Sazerac, 134 (R.F.)
Nakamura, S., 138 (Fev.)
Nakhapetoff, M. I., with Dimow, 65 (Und.)
Namikawa, H., 377 (Mal.)
Nanta, A., 766 (Myc.)
Napier, L. E., 316, 318, 321, 743 (K.A.)
—— & Henderson, J. M., 1036 (Lep.)
——, with Lloyd & Paul, 744 (K.A.)
—— & Mullick, M. N., 321 (K.A.)
Narayanan, R. S., 226 (Rab.)
Nasset, E. C. & Kofoid, C. A., 878 (Am.)
Nataf, R., with Cuenod, 505 (Oph.)
Nathan, H., 557 (Hel.)
Nattan-Larrier, L. & Lépine, P., 202 (S.S.)
——, Ramon, G. & Lépine, P., 208 (S.S.)
Nauck, E. G., 167 (B.R.), 781 (Z.)
Navarro, A. & Spangenberg, J. J., 927 (Mal.)
Navarro-Martín, A., 380 (Mal.)
Nazario, R. C. R., 925 (Mal.)
Nazmi, M., with Augustine, Helmy & McGavran, 553 (Hel.)
——, with Khalil, Peter, El Din & El Betash, 972 (Hel.)
Neff, M. E. A., 1038 (Lep.)
—— & Snodgrass, R. J., 1035, 1036 (Lep.)
Nélis, P., with Bessemans, 133 (R.F.)
Neri, F., 255 (Z.)
Netter, L., 927 (Mal.)
Neuber, E., 985 (Hel.)
Neves, A., 684, 685 (Der.)
Neves, J. S. & Ornelas, A., 363 (Mal.)
Neveu-Lemaire, M., with Brumpt, 434 (B.R.)
—— & Pellegrin, J., 791 (Z.)
Newham, H. B., 703 (S.S.)
—— & Harvey, F. M., 561 (Misc.)
Newman, B. M., with Barber & Komp, 939 (Mal.)
Newman, R. E. U., 485 (Dys.)
Nicholas, M. J., with Cunningham & Lahiri, 219 *ter* (Rab.)
Nicholls, L., 274 (Z.)
Nicholson, D., (993) (Hel.)
Nicolas, Favre & Lebeuf, 862 (C.Bu.)
Nicolas, C., 419 (Misc.), 456 (Fev.)
Nicolas, J., Lebeuf, F. & Rousset, J., 863 (C.Bu.)
Nicolau, S., 218 (Rab.)
——, Dimancesco-Nicolau, O. & Galloway, I. A., 727 (Rab.)
—— & Serbanescu, V., 218 (Rab.)
Nicolle, C., 456 (Fev.)
—— & Anderson, C., 116, 118, 652, 659, 668 (668) *bis* (R.F.), 740 (K.A.)
——, —— & Colas-Belcour, J., 118, 658, 668 (R.F.)
——, —— & Hornus, P., 657 (R.F.)
——, —— & Rahal, M., 657 (R.F.)
——, Mathis, C. & Anderson, C., (128) (R.F.)
Nicolle, P., 119 (R.F.)
Nicolsky, W. W., with Demina, 252 (Z.)
Nigeria, 174 (Y. & S.)
——, Ann. Rep. African Hospital Laboratory, 391 *bis* (Lab.)
——, Ann. Rep. Med. Res. Inst., 390 (Lab.)
Niño, F. L., 475 (Am.), (489) (Dys.), 704, 705 (S.S.)
Nishibe, M., with Ando, 217 (Rab.)
Nissenbaum, B., 934 (Mal.)
van Nitsen, R., 854 (Y. & S.)
——, with Jean, 11 (Mal.)
Nocht, B., 414 (Misc.)
—— & Kikuth, W., 1028 (Bl.)
Noel, P., 111 (Der.)
Noguchi, H., 54, 463 (Fev.)
Noguchi, K., with Matsumura et al, 965 (Bb.)
——, Shannon, R. C., Tilden, E. B. & Tyler, J. R., 849 (Fev.)
Nöller, W., 75 (B.R.)
Nomura, G., with Mikawa & Harada, (950) (Mal.)
Normet, L., Urbain, A. & Chaillot, L., 894 (Dys.)
Notton, H. E. F., with Eyre & Pope, 567 (Misc.)
Nowland, R. E., 46 *bis* (Fev.)
de Nucci, L. S., with Bruchmann, 487 (Dys.)
Núñez, A., with Rickard & Quieta, (950) (Mal.)

## O

Ochi, S., 531 (Hel.)
Ochiai, S., with Matsumura et al, 965 (Bb.)
Ochs, G. W., 560 (Misc.)
O'Connor, F. W. & Burke, G. R., 983 (Hel.)
Oekonomides, G. D., 835 (Fev.)

O'Flynn, J. A., 6 (Mal.)
Office International d'Hygiène Publique, 95 (Pl.)
Ogata, N., Nagai, S. & Unno, S., 53 (Fev.)
Oh, H. Y., 547 (Hel.)
Ohba, T., 556 (Hel.)
Ohmichi, N., 341 (Lep.)
Okell, C. C., with Blake, 482, 889 (Dys.)
Okropiridse, B., with Kandelaki & Abuladse, (949) (Mal.)
Olitzki, L. & Reich, K., 899 (Dys.)
Oliván, N., with Pozzo, (489) (Dys.)
Olivares, R., (600) (Misc.)
de Oliveira, A. B., with Lambert, (950) (Mal.)
Olivier, P. H. & Hulshoff, A. A., 20 (Mal.)
Ollivier, M., with Le Bourdellès & Liégeois, 474 (Am.)
Olmer, D., 458 (Fev.)
——, with Burnet, 49 (Fev.)
——, with —— & Durand, 460 *bis* (Fev.)
—— & Olmer, J., 458 (Fev.)
Olpp, 622 (Lep.)
Ongkiehong, H. F., 902 (Dys.)
—— with Brunnekreeft, 665 (R.F.)
Onorato, R., 666 (R.F.)
Onsy Bey, A., 971 (Hel.)
Orenstein, A. J., 532 (Hel.)
Ornelas, A., with Neves, 363 (Mal.)
Orr, W. B. F., with Sinton & Ahmad, 381 (Mal.)
——, with —— & Bird, 380 (Mal.)
Ortega Corrochano, D., 315 (K.A.)
Otake, M., 551 (Hel.)
Otero, F., 996, (1008) *ter*, (Y.F.)
Otero, P. M., 91 (Pl.)
Ott, 461 (Fev.)
Otto, G. F., with Cort & Spindler, 985 (Hel.)
Otto, R., 778, 779 (Z.)
Ottolenghi, D., with Bonalberti & Brighenti, D., 913 (Mal.)
—— & Brotzu, G., 923, 948 (Mal.)
Ouchakow, W. G., 735 (Rab.)
Oudard, with Dargein, 469 (Am.)
Oudintzew, G. N., with Michailowa, 240 (Z.)
Ouzilleau, P., (824) (Z.)
Owen, D. U., 384 (Mal.), 577 (Misc.)
—— & Murgatroyd, F., 1016 (Bl.)
Owsjannikowa, O. W., with Rosenholz & Trefilow, 113 (R.F.)
Ozawa, M., 536, 972 (Hel.)

## P

di Pace, I., (950) (Mal.)
Pacheco, G., 597 (Misc.)
——, with Fialho & Bicalho, 300 (Y.F.)
Pachêco de Figueiredo, F., with de Mello, (489) (Dys.)
Padua, R. G., 10 (Mal.)
Page, I. H., Turner, K. B. & Wilson, J. H., 592 (Misc.)
Pai, M. N., 462 (Fev.)
Paldrock, A., 1039, 1042 (Lep.)
Palestine, 365 *bis* (Mal.), 403 (Lab.)
Palma, M. D., 21 (Mal.)
Palmer, F. J., 108 (Der.) (632) (Chl.)
Pampana, E. J., 272 (Z.), 585 (Misc.)
——, with Alessandrini, 574, 575 (Misc.)
Panayotatou, (112) (Der.), 328 (K.A.), 803 (Z.), 871 (Am.)
Pani, M., 646 (Y. F.)
Paoletti, U., 386 (Mal.)
Papadopoulo, J., with Petzetakis, 767, 769 (Myc.)
Papadopoulo, T., Yoel, M. & Hadjigeorges, A., 835 (Fev.)
Papanikolau, B., 837 (Fev.)
Pappalardo, C., 149 (Am.), (903) (Dys.)
Paradiso, F., 746 (K.A.)
Parker, W. G., 447 (Und.)
Parmanand, M. J., (676) (Fev.)
Parreiras, D., (642) (Pl.)
——, with Lintz, (1008) (Y.F.)
Parrot, L., 257, 266 (Z.), 748, 754 *ter* (K.A.)
——, with Donatien & Lestoquard, 755 (K.A.)
——, with Sergent, Edm. & Et., 693 (B.R.)
——, with ——, Foley, & Catanei, 362, 363 (Mal.)
——, with ——, ——, —— & Senevet, 3 (Mal.)
Parsons, R. P., 179 (Y. & S.)
Pasqual, J. H., 173 (Y. & S.)
Pasteur, F., with Phisalix, 274 (Z.), 738 (Rab.)
Patané, C., 315 (K.A.)
do Pateo, Jr., J. D., with Gomes, 617 (Lep.)
Paterni, L., (950) *bis* (Mal.), 1019 (Bl.)
Paterson, A. R., (38), (388) (Mal.), 406 (Misc.)
Patterson, S. W., 529 (Hel.)
Paul, B. K., with Megaw & Bhattacharji, 353 (Bb.)
Paul, S. N., with Lloyd, 319 *bis* (K.A.)
——, with —— & Napier, 744 (K.A.)
Pawan, J. L., 237 (Z.), 563 (Misc.)
Pawlowski, E. N., 266 (Z.)
Pawlowsky, E. N., 790 (Z.)
—— & Stein, A. K., 264 (Z.)
—— & ——, with Perfiljew, P. P., 247 (Z.)
Payne, G. C., 553 (Hel.)
Pearse, A. S., 264 (Z.)
Pecori, G. & Escalar, G., 34 (Mal.)
Pedrazzi, A., 723 (S.S.)
Peel, A. A. F., 768 (Myc.)
Peirrier, 591 (Misc.)
Peking Union Medical College, 867 (B.R.)
Pelle, A. & Le Baron, 470 (Am.)
Pellegrin, J., with Neveu-Lemaire, 791 (Z.)
Peltier, M., 617 (Lep.)
Peña, M., with Lozano, 651 (R.F.)
Peña Chavarria, A., 763 (Myc.), (950) (Mal.)
Penfold, W. J. & Corkill, A. B., 52 (Fev.)
Penido, J. C. N., 300 (Y.F.)
Penna, O. & de Figueiredo, B. (1008) (Y.F.)
de Penning, H. C., 12 (Mal.)
Peralta Ramos, A., 336 (Lep.)
Pérard, C., 803 (Z.)
Perekropoff, G. J., 26 (Mal.)
Perekropow, G. J., 29, 946 (Mal.)
Perez Pardo, J., 226 (Rab.)
Perfiljew, P. P., 257 (Z.)
——, with Pawlowsky & Stein, 247 (Z.)
Pergher, G., with Sanarelli, 669 *bis* (R.F.)
Perry, H. M. & Bensted, H. J., 897 (Dys.), 973 (Hel.)
Pershad, J., with Hance, 580 (Misc.)
Peruchena, J. G., 761 (Myc.)
Peruzzi, M., 573 (Misc.), 823 (Z.)
Pervès, 39 (Fev.)
Peryassú, A. G., with Barreto, 650 (Y.F.)

Pessoa, S. B., with Gomes, 685 (Der.)
Peter, F. M., 876 (Am.)
——, with Khalil, Nazmi, El Din & El Betash, 972 (Hel.)
Peterson, D. H., 794 (Z.)
Peterson, R. A., 506 (Opk.)
Petridis, 871 (Am.)
Petridis, P., 591 (Misc.)
Petrov, V. P., 1043 (Lep.)
Pettit, A., 1008 (Y.F.)
—— & Stefanopoulo, G., 308 (Y.F.)
——, —— & Frasey, V., 307 (Y.F.)
——, —— & Kolochine, C., 1007 (Y.F.)
Petty, W. S., 919 (Mal.)
Petzetakis, M., 468 (Am.), 578 (Misc.), (903) (Dys.)
—— & Papadopoulo, J., 767, 769 (Myc.)
Peterson, R. A., 506 (Oph.)
Peverelli, P., 85, 627, 632 *bis* (Chl.), 528, 969 (Hel.)
Pevsner, A. S., (950) (Mal.)
Pezzi, G., 942 (Mal.)
Pfannenstiel, W. & Scharlau, B., 709 (S.S.)
Phease, R. N., 847 (Fev.)
Phelps, B. M., 16 (Mal.), 271 (Z.)
Phelps, E. B. & Dávila, J. V., 162 (Dys.)
Philip, C. B. (258) (Z.), 1005 (Y.F.)
——, with Hudson & Bauer, 647 (Y.F.)
——, with —— & Davis, 1005 (Y.F.)
Phillips, W., 635 (Pl.)
Philiptschenko, A., 712 (S.S.)
Philpott, C. H., 793 (Z.)
Phisalix, 739 (Rab.), 780 (Z.)
—— & Pasteur, F., 274 (Z.), 738 (Rab.)
Phòtakis, B. A., 926, (950) (Mal.)
Photinos, T., 687 (Der.)
Picado, C., 942 (Mal.)
Picard, F., 9 (Mal.)
Pichat, J., 649 (Y.F.)
Pickoul, I. N., 651 (R.F.)
Pickoul, J. N., with Marzinowsky & Balaschewa, 373 (Mal.)
Pieri, J., with Boinet & Dunan, 49, 459 (*bis*) (Fev.)
Pierini, L. E., 338, 343 (Lep.)
Pigulewsky, S. W., 530 (Hel.)
Pillai, K. V., with Chopra & Gupta, 564 (Misc.)
Pimentel, C., 970 (Hel.)
Pin, M., with Blanchard, 948 (Mal.)
Pinelli, L., (950) (Mal.)
Pinto, G. de S., 10 (Mal.)
—— & Pinto, S. F., 369 (Mal.)
Pinto, W., 968 (Bb.)
Pirami, E., (489) (Dys.)
Pires, W. & Póvoa, H., 31 (Mal.)
Pirker, H., with Moritsch, 274 (Z.)
Pirot, R., with Morin, 455 (Fev.)
Pittaluga, Martinez Cepa, Torrijos & Ginés, 917 (Mal.)
Pittaluga, G., (309) (Y.F.)
Plaut, F., 113 (R.F.)
Plavtov, K., 973 (Hel.)
Plazy, Marcandier & Marcon, 457 (Fev.)
—— & Marçon, 457 (Fev.)
Plehn, A., 25 (Mal.)
Plochmann, E., with Leuchs, 478 (Dys.)
Poddighe, A., 446 (Und.)
Podyapolskaya, V. P., with Scriabine & Schoultz, (993) (Hel.)
Poggi, I., (676), 842 (Fev.)
Poldie, H., 122 (R.F.)
Pomaret, with Lavall, 340 (Lep.)
Ponomareff, A. W. & Solovieff, N. N., 732 (Rab.)
Ponomarew, A. W., 481 (Dys.), 738 (Rab.)
—— & Soloview, N. N., 731 (Rab.)
Pons-Leychard, A., 328 (K.A.)
Pontano, T., 452 (Fev.)
del Ponte, E., with Shannon, 248 (Z.)
v. Poór, 357 (Pel.)
ter Poorten, F. H., 870 (Am.)
Pope, W. J., with Eyre & Notton, 567 (Misc.)
Popow, P., with Zeiss, 490 (Sp.)
Poppe, K., 442 (Und.)
Porter, A., 525 (Hel.), 824 (Z.)
Portocalis, A. & Flora, G., 453 (Fev.)
Portois, 171 (Y. & S.)
Porto Rico Review of Public Health & Tropical Medicine, 1 *bis*, 27, 366 (Mal.)
Poumailloux, M., with Aubertin, 671 (R.F.)
Poumeau-Delille, G., with Laederich, 880 (Am.)
Póvoa, H., with Pires, 31 (Mal.)
Pozzi, A., 28 (Mal.)
Pozzo, F. & Oliván, N., (489) (Dys.)
Prado, A., 21 (Mal.)
Prigge, R. & Rothermundt, M., 114 (R.F.)
di Primio, R., (950) *bis* (Mal.)
Pritze, F., 239 (Z.)
Proute, 369 (Mal.)
Proutowych, V. O., (330) (K.A.)
Provera, G., 290 (Hist.)
Provera, P., (824) (Z.)
Pruthi, H. S., 246 (Z.)
Przesmycki, F., Lipowska, L. & Sierakowski, S., 157 (Dys.)
Public Health Reports, 56 (Und.), 609 (Lep.), 994 (Y.F.)
Puchulu, F., 873 (Am.)
Puente, J. J., 610, 1034 (Lep.)
Puff, G., 685 (Der.)
Puntoni, V., 222 (Rab.)
Puri, I. M., 250, (258) (Z.)
Puyal, J., 380 (Mal.)

## Q

Quieta, P. D., with Rickard & Núñez, (950) (Mal.)
Quintini, J., 788 (Z.)

## R

Rabbani, S. M., 13 (Mal.)
Radsma, W., 578 (Misc.)
—— & Joenoes, M. I., 578 (Misc.)
Raffaele, G., 241 (Z.)
——, with La Face, 254 (Z.)
Ragazzi, C. A., (642) (Pl.)
Raghava, S. N., (38) (Mal.)
Ragu, with Brulé & Laporte, 880 (Am.)
Rahal, M., with Nicolle & Anderson, 657 (R.F.)
Raimondi, S. & Canal Feijóo, E. J., 121 (R.F.)
Raiziss, G. W. & Severac, M., 212 (S.S.)
Ramon, G., with Nattan-Larrier & Lépine, 208 (S.S.)

Ramon Giner, D., 704 (S.S.)
Ramos e Silva, J., 103, 111 (Der.)
Ramos Baez, P., with Hoffmann, (1044) (Lep.)
Ramsay, G. W. St. C., 334 (Lep.)
Ramsine, S., with Konieff, 221 (Rab.)
Ranganathan, K. S., 100 (Pl.)
Rangel, M., 616 (Lep.)
Ranieri, G., 977 (Hel.)
Ranjeva, 1030 (Bl.)
Rao, G. R., 368 (Mal.), 599 (Misc.)
Rao, M. G. R., 108 (Der.)
Rao, S. S., 545 (Hel.), 846 (Fev.)
——, with Chopra, 984 (Hel.)
——, with Megaw, 49 (Fev.)
Ratcliffe, H. L., 234 (Z.), 381 (Mal.)
Raúl Goyena, J., (165) (Dys.)
Rault, with Voizard, 666 (R.F.)
Raynal, J., 387, 934 (Mal.), 526 (Hel.)
—— & Leger, J., 556 (Hel.)
Raynard, L. & Colonieu, L., 328 (K.A.)
Razafindramamba, with Legendre & Mondain, 949 (Mal.)
Redaelli, P., 759 (Myc.)
Reed, A. C., 924 (Mal.)
Reeder, J. E., 506 (Oph.)
Rees, C., 796 (Z.)
Rees, C. W., 473 (Am.)
Reeves, I. S. K., 344 (Lep.)
Regendanz, P., 126 (R.F.), 721, 722 (S.S.), 884 (Am.), (1008) (Y.F.)
—— & Kikuth, W., 795 (Z.)
Register, J. C., 105 (Der.)
Reich, K., with Olitzki, 899 (Dys.)
Reichenow, E., 692 (B.R.)
Reimann, H. A., with Hu & Kurotchkin, 765 (Myc.)
——, Kurotchkin, T. G. & Tso, E., 765 (Myc.)
Reiss, F., 1033 (Lep.)
Reitani, U., with Chiabrera, 52 (Fev.)
Reitler, R., 382 (Mal.)
——, with Kligler, 364, 922 (Mal.)
—— & Saliternik, H., 811 (Z.)
Remlinger, P., 140 (Am.), 217, 725 (Rab.), 335, (625) (Lep.)
—— & Bailly, J., 218, 222, 224, 736 *bis*, 737 *bis* (Rab.)
Renaud, M., 274 (Z.)
República Argentina, 605 (B.R.)
Revue de Médecine et d'Hygiène Tropicales, 871 (Am.)
Rey, G. B., with Bejarano & Abadia, (37) (Mal.)
de Rezende, C., (309) (Y.F.)
Rheindorf, G., with Fischer, 376 (Mal.)
Ribeiro da Fonseca, O. O., 762 (Myc.)
Rice, J. P., with Williams & Lacayo, 284 (Hist.)
Rice, T. B., 728 (Rab.)
Richardson, W. & Klumpp, T. G., 495 (Sp.)
Richter, O., Maurer, S. & Eyl, M., 540 (Hel.)
Rickard, E. R., 815 (Z.)
——, with Davis, (949) (Mal.)
——, with Mazza, 366 (Mal.)
——, Quieta, P. D. & Núñez, A., (950) (Mal.)
Ricklin, J., with Fourche, 193 (S.S.)
Ricou, D., 596 (Misc.)
Riddell, W., 557 (Hel.)
Ridley, F., 508 (Oph.)
van Riel, 410 (Misc.)
van Riemsdijk, M., 87 (Chl.)
da Rin, O. & Weinberger, M., (74) (Und.)
Riqueau, 807 (Z.).
de Rivas, D., 789 (Z.)
—— & Fife, C. A., 527 (Hel.), 787 (Z.)
Robbins, F. C., 946 (Mal.)
Robbins, J. H., 361 (Mal.)
Roberts, F. L., with Meleney & Bishop, 919 (Mal.)
Robertson, A., with Thomson, 435 (B.R.)
Robic, J., 479 (Dys.)
——, with Girard, 861 (Y. & S.)
Robineau, 615 (Lep.)
Robinson, F. E., 383 (Mal.)
Roch, M., 820 (Z.)
——, Monedjikova, V. & Martin, E., 63 (Und.)
Rockefeller Foundation, 954 (B.R.)
Rodenwaldt, E., 32 (Mal.)
Rodhain, J., 110, 688 (Der.)
Rodriguez, J., 336 (Lep.)
Rodriguez, R., with Casas & de Buen, 314 (K.A.)
Rodriguez Oliva, R., 21 (Mal.)
Roffo, A. H., 1043 (Lep.)
Rogers, L., 83 (Chl.), 633 (Pl.), 953 (B.R.
Romiti, C., 599 (Misc.)
Ronnefeldt, F., (950) (Mal.)
Roppongi, H., 777 (Z.)
Roques, P., (642) (Pl.)
Rose, F. G., 621 *bis* (Lep.)
Rosenau, M. J., 226 (Rab.)
Rosenbaum, S., (903) (Dys.)
Rosenberg, M., 842 (Fev.)
Rosenblatt, A., with Lurje & Kossarew, 891 (Dys.)
Rosengolz, H. P., with Stern, 207 (S.S.)
Rosenholz, H. P., Owsjannikowa, O. W. & Trefilow, I. A., 113 (R.F.)
Roskin, G. & Schischliaiewa, S., 799 (Z.)
Roskott, E. R. A. L., with Bonne, 927 (Mal.)
—— & Séno, R., 20 (Mal.)
Ross, P., 947 (Mal.)
Ross, R., (38), 914, 924 (Mal.), 166, 427, 956 (B.R.), 807 (Z.)
Ross, W. C., 313 (K.A.)
Rossi, G., 914 (951) (Mal.)
Rothermundt, M., with Hartoch, 662 (R.F.)
——, with Prigge, 114 (R.F.)
Rotta, C., with Bastai, 68 (Und.)
Roubaud, E., 95 (Pl.), 256, 808 (Z.)
Rousset, J., with Nicolas & Lebeuf, 863 (C.Bu.)
Row, R., 381, 383, 935 *bis* (Mal.)
Rowntree, L. G., with Hunt, McCann, Voegtlin & Eggleston, 378 (Mal.)
Roy, A. C., with Boyd, 330 (K.A.)
Roy, D. N., 686 (Der.)
Ruchadse, N., (951) *bis* (Mal.)
Rudolf, G. de M., 935 (Mal.)
Rudolfs, W. & Lackey, J. B., 806 (Z.)
Ruge, H., 115 *bis*, 128, 135 (R.F.), 266 (Z.)
Ruge, R., 9 (Mal.), 1009 (Bl.)
Russell, P. F., 8 (Mal.)
Russian Socialist Federal Soviet Republic, 36 (Mal.)
Ruttledge, W., 819 (Z.)
Ruys, A. C., with Kuipers, 674 (Fev.)
Ryrie, G. A., 550 (Hel.)

S

Sabatucci, M., 942 (Mal.)
Sabrazès, J., 128 (R.F.)
Sachs, H. & Klopstock, A., 541 (Hel.)
Sacón, J. J., 276 (Z.)
Saenz, A., with Blanc, Caminopetros & Dumas, 838 (Fev.)
Sagel, W., 654 (R.F.)
Sainte-Marie, P. E. F., 240 (Z.)
Sakaguchi, T., 550, 551 (Hel.)
Sakurai, H., (344) (Lep.)
Salas, L. M., with Iriarte, 864 (G.V.)
Saliternik, H., with Reitler, 811 (Z.)
Salle, A. J. & Schmidt, C. L. A., 329 (K.A.)
Salminen, Y. W., 618 (Lep.)
Salmon, W. D., Hays, I. M. & Guerrant, N. B., 359 (Pel.)
Salvioli, G., 158 (Dys.)
Salzberger, M., 609 (Lep.)
Samson, J. G. & Lara, C. B., 1036 (Lep.)
Sanarelli, G., 131 (R.F.)
—— & Pergher, G., 669 *bis* (R.F.)
Sanchis-Bayarri, V., 222 (Rab.)
Sanders, E. P., 883 (Am.)
Sanders, H. C., 257 (Z.)
Sanderson, E. S., with Smith & Turner, 763 (Myc.)
Sandground, J. H., 971 (Hel.)
Sandu, D. V., 82 (B.R.)
Sanfilippo, E., 830 (Und.), 875 (Am.)
——, with Ascoli, 68, 830 (Und.)
—— & Migneco, G., 445 (Und.)
Sanford, M. F., with Light, 790 (Z.)
Santesson, C. G., 598 (Misc.)
Santos, J., (309) (Y.F.),
Sarcar, S. L., 5 (Mal.)
Sarcinella, F., (558) (Hel.)
Sarcorrafos, M., 451 (Fev.)
Sardjito, M., with Walch, 251 *bis* (Z.)
——, M. & Zuelzer, M., 670 (R.F.)
Sartori, C., 444 (Und.)
Sartorius, F., 886 (Dys.)
Sartory, A. & R., Marcel & Mayer, J., 683 (Der.)
Sasaki, R., 477 (Dys.)
Sato, A., 562 (Misc.)
Sato, T., with Matsumura et al, 965 (Bb.)
—— & Shoji, K., 561 (Misc.)
Satow, T., 1043 (Lep.)
Saujeon, M., 471 (Am.)
Saunders, A. M., with Hershfield, Kibler, Colby, Koenig & Schmid, 675 (Fev.)
Saunders, G. F. T., 708 (S.S.)
Sautet, J., 166 (B.R.), 475, 886 (Am.)
——, with Horovitz, 936 (Mal.)
Savas, C. & Cardamatis, J. P., 428 (B.R.)
Savorini, G., with Guigni, 57 (Und.)
Sawyer, W. A., Lloyd, W. D. M. & Kitchen, S. F., 1007 (Y.F.)
Sayers, E. G., 369 (Mal.)
Sazerac, R. & Nakamura, H., 134 (R.F.)
—— & Vaurs, R., 127 (R.F.)
Schaechter, E. P., with Aristowsky, 127 (R.F.)
Schafir, M. M., 542 (Hel.)
Schapiro, S., with Kritschewski, 420 (Misc.)
Scharff, J. W., 33 (Mal.)
Scharlau, B., with Pfannenstiel, 709 (S.S.)
Schaumann, O., 876 (Am.)
Schern, K., 722 (S.S.), 738 (Rab.)
Schewelewa, E. M., with Wolski, 927, 938 (Mal.)
Schewtschenko, F. I., 256 (Z.)
——, with Chodukin, 323 (K.A.)
Schéwtschénko, P. J., with Soffiewe, 755 (K.A.)
Schiassi, F. & Merighi, G., 19 (Mal.)
Schill, E., 488 (Dys.)
Schilling, C., 721 (S.S.), 916 (951) (Mal.)
—— & Sollazzo, G., 720 (S.S.)
Schilling, V., 524 *bis* (Z.)
Schirwindt, S. L., 618 (Lep.)
Schischliaiewa, S., with Roskin, 799 (Z.)
Schlossberger, H., 138 (Fev.)
—— & Wichmann, F. W., 664 (R.F.)
Schlossmann, K., 1042 (Lep.)
Schmaus, L. F., 821 (Z.)
Schmid, O. W., with Hershfield, Kibler, Colby, Koenig & Saunders, 675 (Fev.)
Schmidt, C. L. A., with Salle, 329 (K.A.)
Schmidt, H., 569 (Misc.)
Schmidth, F. J. W., 13 (Mal.)
Schmourlo, S., 39 (Fev.)
Schneider, J., 947 (Mal.)
Schnitzer, R. & Silberstein, W., 203 (S.S.)
Schöbl, 172 (Y. & S.)
Schöbl, O. & Villaamil, R., 155 (Dys.)
Schockaert, J., 119 (R.F.), 135 (Fev.)
Schoultz, R. S., with Scriabine & Podyapolskaya, (993) (Hel.)
Schourenkowa, A. I. & Lissovsky, L. L., 232 (Z.)
Schoute, E., with de Buck & Swellengrebel, (824) (Z.), 915 (Mal.)
Schrumpf-Pierron, P., 840 (Fev.), 872 (Am.)
Schuchat, J. A., with Gromaschewskij, J. A., 268 (Z.)
Schüffner, W., 25 *bis* (Mal.)
Schüffner, W. A. P., with Dinger, Snijders & Swellengrebel, 1006 (Y.F.)
Schule, P. A., 43 (Fev.)
Schulz, R. E., 990 (Hel.)
Schuurman, C. J. & Huinink, A. S. B., 912 (Mal.)
Schwarzmann, B., 1034 (Lep.)
Schwarzmann, L., 675 (Fev.)
——, with Kritschewski, 420 (Misc.)
Schweinburg, F., 216 (Rab.)
——, with Gerlach, 726 (Rab.)
Schweizerische Medizinische Wochenschrift, 986 (Hel.)
Schwetz, J., 249 (Z.)
——, with Collart, A. & Geernick, 939 (Mal.)
Scordombekis, E., with Chaniotis, 836 (Fev.)
Scott, G. H., with Cowdry, 239 (Z.)
Scott, J. A., 992 (Hel.)
Scozzafave, I. & Warner, W. P., 63 (Und.)
Scriabine, K. I., Podyapolskaya, V. P. & Schoultz, R. S. (993) (Hel.)
Scuderi, G., 146 (Am.)
—— & Baroni, B., 880 (Am.)
Seagar, E. A., 1 *bis* (Mal.)
Sébenzow, B. M., with Smorodinzew & Adowa, 812 (Z.)
Sechi, E., 576 (Misc.)
Secques, F., 857 (Y. & S.)
Seelemann, M. & Hadenfeldt, A., 443 (Und.)
Segal, M. B., with Fried, 679 (Der.)
Seiffert, with Uhlenhuth, 672 (R.F.)
Seki, T., with Matsumoto, 481 (Dys.)

Seki, Y., 489 (Dys.)
Sellards, A. W. & Mathis, C., 647 (Y.F.)
—— & Siler, J. F., 43 (Fev.)
——, with Theiler, 306 (Y. F.)
Selwyn-Clarke, P. S., 644 (Y.F.)
Semikoz, F., 641 (Pl.)
—— & Lokhov, M., 641 (Pl.)
Seminario, C. & Gaviña Alvarado, E. R., (1044) (Lep.)
Sen, K. C., with Dhar, 87 (Chl.)
Sen, P. B., with Brahmachari, 559 (Misc.)
Sen, R. T., with Hodgson, 322 (K.A.)
——, with —— & Das, 321 (K.A.)
Senevet, G., with Sergent, Edm. & Et., Parrot, Foley & Catanei, 3 (Mal.)
Senior-White, R., 245, 246 (Z.)
Séno, R., with Roskott, 20 (Mal.)
Sensenich, R. L. & Giordano, A. S., 60 (Und.)
Sepaschwili, A., (951) (Mal.)
Sepulcri, P. & Vidale, E., 949 (Mal.)
Sequeira, J. H., 108 (Der.)
Serbanescu, V., with Nicolau, 218 (Rab.)
Serefis, S., with Habermann, (388) (Mal.)
——, with Keining, 385 (Mal.)
Sergent, Edm., 397 (Lab.)
Sergent, Edm. & Sergent, Et., 946 (Mal.)
——, —— & Parrot, L., 693 (B.R.)
——, ——, Foley, H. & Catanei, A., 362, 363 (Mal.)
——, ——, ——, —— & Senevet, G. 3 (Mal.)
Sergi, A., (388) (Mal.)
Serra, A., 220 (Rab.), 492 (Sp.)
Servidori, G., (600) (Misc.)
Seshachalam, T., (600) (Misc.)
Sette, N., (951) (Mal.)
Severac, M., with Raiziss, 212 (S.S.)
de Sèze, S., with Tixier, 134, 672 (R.F.)
Shaha, B., 22 (Mal.)
Shannon, R. C., with Davis, 253 (Z.) 1000 (Y.F.)
——, with Noguchi, Tilden & Tyler, 849 (Fev.)
—— & del Ponte, E., 248 (Z.)
Sharp, W. B. & Taylor, E. K., 680 (Der.)
Sharpe, W. S., 856 (Y. & S.)
Shattuck, G. C., 350 (Bb.) (600 *bis*) (Misc.)
Shaw, Jr., E. H., 24 (Mal.)
——, with Hegner & Manwell, R. D., 24 (Mal.)
Shaxby, J. H. & Bonnell, H. E., 576 (Misc.)
Shelmire, B., 555 (Hel.)
Shiozawa, S., 130 *bis* (R.F.)
Shirai, M., 529 (Hel.)
Shiraiwa, T., with Matsumoto & Ando, 87 (Chl.)
Shircore, J. O., (724) (S.S.)
Shoji, K., with Sato, 561 (Misc.)
Shortt, H. E., Craighead, A. C., Smith, R. O. A. & Swaminath, C. S., 322, 323 (K.A.)
——, —— & Swaminath, C. S., 310 (K.A.)
——, d'Silva, H. A. H. & Swaminath, C. S., 325 (K.A.)
—— & Swaminath, C. S., 236 (Z.)
Shousha, A. T. & Aly, M., 524 (Z.)
Shu, H. J., 564 (Misc.)
Shvarcman, M., (388) (Mal.)
Sicé, A. & Vaucel, M., 813 (Z.)
Sierakowski, S., with Przesmycki & Lipowska, 157 (Dys.)
Silberstern, E., (824) (Z.)
Silberstein, E., 667 (R.F.), 795 (Z.)
Silberstein, W., with Schnitzer, 203 (S.S.)
Siler, J. F., with Sellards, 43 (Fev.)
Silva, with d'Utra, 750 (K.A.)
Silva, F., (359) (Pel.), 763 (Myc.)
da Silva, F. V., 856 (Y. & S.)
d'Silva, H. A. H., with Shortt & Swaminath, 325 (K.A.)
da Silva, J. R., (600) (Misc.)
da Silva, R., 138 (Fev.)
Silveira, F. & Gomes, J. M., 1041 *bis* (Lep.)
Silvestro, L., 683 (Der.)
Simanin, P., 28 (Mal.)
Simanine, P. I., with Smirnowa, 28 (Mal.)
Simmons, J. S. & St. John, J. H., (951) (Mal.)
——, with —— & Gardner, 988 (Hel.)
Simmons, R. J., 819 (Z.)
Simonetti, F., 71 (Und.)
Sims, L. C., with Gill & Kiker, 919 (Mal.)
Sinclair, M. J., 553 (Hel.)
Siniscalchi, R., with Missiroli, (950) (Mal.)
Sinton, J. A., 255, 256, 815 (Z.)
—— & Barraud, P. J., 255 (Z.)
—— & Bird, W., 14, 929 (Mal.)
——, —— & Eate, S. N., 379 (Mal.)
——, —— & Orr, W. B. F., 380 (Mal.)
——, with Christophers & Covell, 360 (Mal.)
——, Orr, W. B. F. & Ahmad, B., 381 (Mal.)
Sivamani, V., 13 (Mal.)
Sivasambandan, R., 550 (Hel.)
Skorodoumoff, 641 *bis* (Pl.)
Skorodumow, A., 637 (Pl.)
Slanetz, C. A., with McAlpine, 74 (Und.)
de Smidt, F. P. G., 99 (Pl.)
Smirnov, G., 969 (Hel.)
Smirnowa, A. M., with Adowa, 814 (Z.)
Smirnowa, E. W. & Simanine, P. I., 28 (Mal.)
Smith, D. C., Turner, H. C. & Sanderson, E. S., 763 (Myc.)
Smith, E. C., 104, 107 (Der.)
—— & Elmes, B. G. T., 688 (Der.)
Smith, H., 507 (Oph.)
——, with Strickland, Chowdhury, Dodds-Price, Forsyth, Murphy & Williams, 4 (Mal.)
Smith, H. S., 686 (Der.)
Smith, M., with Isaacs & Sturgis, 540 (Hel.)
Smith, N. M. & Barret, H. P., 231 (Z.)
Smith, R. O. A., with Shortt, Craighead & Swaminath, 322, 323 (K.A.)
Smith, S., 846 (Fev.)
Smithies, F., 228 *bis* (Z.)
Smits, E., with Baermann, 372 (Mal.)
Smorodinzew, I. A. & Adowa, A. N., 814 (Z.)
——, Sébenzow, B. M. & Adowa, A. N., 812 (Z.)
Snijders, E. P., with Dinger, Schüffner & Swellengrebel, 1006 (Y.F.)
Snodgrass, R. J., with Neff, 1035, 1036 (Lep.)
Soares, P., (46) (Fev.)
Société Médico-Chirurgicale de l'Ouest-Africain Français, (600) (Misc.)
Soesilo, R., 812 (Z.), 912 *bis*, 928 (Mal.)
——, with Walch, 911 (Mal.)
Soffieff, M. S., with Chodukine, 324 (K.A.)
Soffiewe, M. S. & Schéwtschénko, P. J., 755 (K.A.)
Solares, A., 505 (Oph.)
Soliani, G., 282 (Hist.)
Sollazzo, G., 714 (S.S.)
——, with Schilling, 720 (S.S.)

Sollini, A., 854 (Y. & S.)
Solovieff, N. N., with Ponomareff, 732 (Rab.)
Soloview, N. N., with Ponomarew, 731 (Rab.)
Sorge, G., 929 (Mal.)
Soru, E., 88 (Chl.)
Souchard, L., 632 (Chl.)
South African Institute for Medical Research, 393 (Lab.)
Southern Medical Journal, 7, 919 (Mal.)
de Souza-Araujo, H. C., (1044) (Lep.)
Spangenberg, J. J., with Navarro, 927 (Mal.)
Sparrow, H., with Lapidari, 120 (R.F.)
——, Lumbroso, U. & Lapidari, M., 665 (R.F.)
Speedy, W. D., (951) (Mal.)
Spillmann, (344) (Lep.)
Spindler, L. A., with Cort & Otto, 985 (Hel.)
Spittel, R. L. & Fernando, S. E., 690 (Der.)
Splatt, B., with Fairley, 773 (Z.)
Spring, D., 764 (Myc.)
Spyropoulos, N. J., 316 (K.A.)
Stanhope, R. A. B., 736 (Rab.)
Stanziale, R., 337, 340 *bis* (Lep.)
Statirova, N. A., with Gnedina, (558) (Hel.)
van Steenis, P. B., 26 (Mal.), 589 (Misc.)
Stéfanopoulo, G. J. & Hosoya, S., 129 (R.F.)
——, G. J., with ——, 483 *bis* (Dys.)
——, with Pettit, 308 (Y.F.)
——, with —— & Frasey, 307 (Y.F.)
——, with —— & Kolochine, 1007 (Y.F.)
Stein, A. A., 1043 (Lep.)
Stein, A. K., with Pawlowsky, 264 (Z.)
——, with —— & Perfiljew, 247 (Z.)
Steiner, G. & Fischl, V., 653 (R.F.)
Steinert, K., 541 (Hel.)
Sterckx, F., 1024 (Bl.)
Stephens, J. R. C., 268 (Z.)
Stephens, J. W. W., 414 (Misc.), 1011 *ter* (Bl.)
Stern, E., (489) (Dys.), 883 (Am.), 932 (Mal.)
Stern, L. & Rosengolz, H. P., 207 (S.S.)
Steudel, 190 (S.S.)
Stévenel, R., 1038 (Lep.)
Stewart, M. A., 818 (Z.)
Stewart, R. C., 138 (Fev.)
Stiles, C. W. & Baker, C. E., 557 (Hel.)
—— & Hassall, A., 241 (Z.)
Stitt, E. R., 515 (B.R.)
Stiven, H. E. S., 590 (Misc.)
St. John, J. H., 31 (Mal.)
——, with Simmons, (951) (Mal.)
——, —— & Gardner, L. L., 988 (Hel.)
Stoll, H. F., 987 (Hel.)
Stovall, W. D. & Greeley, H. P., 761 (Myc.)
Stratman-Thomas, W. K. & Loevenhart, A. S., 198, 199 (S.S.)
Street, A., 146 (Am.)
Strickland, C., assisted by Choudhury, K. L. & others, 909 (Mal.)
——, with ——, Dodds-Price, J., Forsyth, C. E. P., Smith, H., Murphy, R. A. & Williams, D. P., 4 (Mal.)
Ströman, R., with Naeslund, 91 (Pl.)
Strong, W. M., 112 (Der.)
Strozzi, T., with Dagnini, (668) (R.F.)
Struthers, E. B., 13 (Mal.)
Stuart, G. & Krikorian, K. S., 223, 732 (Rab.)
Stuart, H. C. & Augustine, D. L., 542 (Hel.)
Stunkard, H. W., (993) *bis* (Hel.)
—— & Willey, C. H., 974 (Hel.)
Sturgis, C. C., with Isaacs & Smith, 540 (Hel.)
Sudan, 700 (S.S.)
Sülk, N., 622 (Lep.)
Sur, P., 8 (Mal.)
——, with Iyengar, 3 (Mal.)
Surraco, N., with Talice, 665 (R.F.)
Suzaki, K., 484, 485 *bis* (Dys.)
Suzuki, M., with Matsumura et al, 965 (Bb.)
Suzuki, S., 119 (R.F.)
Suzzi, D., 467 (Am.)
Svensson, R., 227 (Z.)
Swaminath, C. S., with Shortt, 236 (Z.)
——, with —— & Craighead, 310 (K.A.)
——, with ——, —— & Smith, 322, 323 (K.A.)
——, with —— & d'Silva, 325 (K.A.)
Sweeney, M. A., with Walker, 1032 (Lep.)
Swellengrebel, N. H., with de Buck & Schoute, (824) (Z.), 915 (Mal.)
——, with Dinger, Schüffner, & Snijders, 1006 (Y.F.)
——, with Honig., 282 (Hist.)
Sydenstricker, E., with Goldberger, Wheeler, King & others, 356, 963 (Pel.)
Symes, C. B., 248 (Z.)
Syphantos, S., 455 (Fev.)
Syssine, A., 636 (Pl.)

## T

Taddia, L., with Bacchelli, (258) (Z.)
Takabashi, S., 108 (Der.)
Takaya, J., 220 (Rab.)
Takeda, S., (165), (489) (Dys.)
Talaat, A., 327 (K.A.)
Taliaferro, L. G., 800 (Z.)
Taliaferro, W. H., 231, 794, 802 (Z.), (388) (Mal.)
——, Hoffman, W. A. & Cook, D. H., 534, 535 (Hel.)
—— & Taliaferro, L. G., 27 (Mal.)
Talianidis, P. P., 1020 (Bl.)
Talice, R., 656 (R.F.)
Talice, R. V., 659 (R.F.)
—— & Mackinnon, J. E., 761 (Myc.)
—— & Surraco, N., 665 (R.F.)
Tamiya, T., 617 (Lep.)
Tanabe, B., 855 (Y. & S.)
Tanabe, M. & Chiba, E., 474 (Am.)
Tanew, I. & Haschnow, G., 933 (Mal.)
Tanikawa, K., with Matsumura et al, 965 (Bb.)
Tapie, J., with Morel, 873 (Am.)
Tarassow, S. I., 673 (R.F.)
Tate, D. L., 13 (Mal.)
Taylor, A. W., 799 (Z.)
Taylor, E. K., with Sharp, 680 (Der.)
Taylor, J. & Thant, U., 966 (Bb.)
Tchernenko, A., with Le Boucher, Capéran & Laigret, 997 (Y.F.)
Tegoni, G., with Frugiuele, T. & Williams, L., (38) (Mal.)
Teiryo, T., with Masataka (344) *quat.* (Lep.)
Teitel, D., 876 (Am.)
Teixeira, J. C., with Villela, 992 (Hel.)
Teodorascu, C., 725 (Rab.)
Teodosio, V., with Jonesco, 728 (Rab.)
Thakkar, K. V., (951) (Mal.)
Thant, U., with Taylor, 966 (Bb.)
Theiler, M. & Sellards, A. W., 306 (Y.F.)
Theodor, O., with Adler, 256 (Z.), 324, 748, 749, 756 (K.A.)
van Thiel, P. H., 815 (Z.)

Thierfelder, M. U., 183 (G.V.)
Thiry, U., with Bessemans, 671 (R.F.)
Thomson, J. G., 908 (Mal.)
—— & Robertson, A., 435 (B.R.)
Thomson, J. W., 1015 (Bl.)
Thonnard-Neumann, E., 12 (Mal.)
Thorlakson, P. H. T., 487 (Dys.)
Tilden, E. B., with Noguchi, Shannon & Tyler, 849 (Fev.)
Timpano, P., 743 (K.A.)
Tisseuil, 611 (Lep.)
Tisseuil, G., 334 (Lep.)
Tisseuil, J., 1033 (Lep.)
Tixier, L. & de Sèze, S., 134, 672 (R.F.)
Todokoro, K., 535 (Hel.)
Tohyama, Y., 133 (R.F.)
Tomb, J. W., 626 (Chl.)
Tomlinson, C. C. & Bancroft, P., 765 (Myc.)
Tonelli, L., 147 (Am.)
Tonietti, F., 422 (Misc.)
Torikata, R. & Uyeda, O., 630 (Chl.)
Török, G., (903) (Dys.)
Torres, C. M., 203 (S.S.), 301 *bis*, 302 (*sex*), 648, (650), 997 *bis* (Y.F.)
—— & de Azevedo, A. P., 299 (Y.F.)
Torres, O., 595 (Misc.)
Torres, S., 731 (Rab.)
Torrijos, with Pittaluga, Martinez Cepa & Ginés, 917 (Mal.)
Toullec, F., 798 (Z.)
—— & Blanchard, M., 829 (Und.), 882 (Am.)
Tournier, E. & Guénolé, A., 455 (Fev.)
Trabaud, (165) (Dys.), 871 (Am.)
—— & Sabbagh, 871 (Am.)
Trabaud, J., 350 (Bb.), (600) (Misc.), 843 (Fev.)
Transactions of the 24th Annual Conference of State and Territorial Health Officers with the United States Public Health Service, 225 (Rab.)
Transactions of the Royal Society of Tropical Medicine & Hygiene, 405 (Lab.)
Trautmann, E., (388) (Mal.)
Trefilow, I. A., with Rosenholz & Owsjannikowa, 113 (R.F.)
Trelles, R., with Mazza, (38) (Mal.)
Trensz, F., 86 (Chl.)
Trevan, J. W., 482 (Dys.)
Tripoli, C. J., 885 (Am.)
Troise, E., 278 *bis* (Z.)
Troisier, J., Deschiens, R., Limousin, H. & Delorme, M., 556 (Hel.)
Trolli, G., 698 (S.S.)
Truffi, G., 1039 (Lep.)
Trujillo, with Carrancá, 963 (Pel.)
Tsatsaronis, with Bourgeois, 339 (Lep.)
Tsen, E. T. H. & Chi, C. C., 735 (Rab.)
Tso, E., with Reimann & Kurotchkin, 765 (Myc.)
Tsu, T. Y., 237 (Z.)
Tsuchiya, K. & Chuan, L. T., 634 (Pl.)
—— & Nagata, S., 487 (Dys.)
Tsurumi, M., (642) (Pl.)
Tubangui, M. A., 531 *bis* (Hel.)
Tull, J. C., 85 (Chl.), 351 (Bb.)
Turkhud, D. A., (135) (R.F.)
Turner, H. C., with Smith & Sanderson, 763 (Myc.)
Turner, K. B., with Page & Wilson, 592 (Misc.)
Turner, R. H., 960 (Pel.)
Tuxford, A. S., 352 (Bb.)
Tyler, J. R., with Noguchi, Shannon & Tilden, 849 (Fev.)

## U

Uhlenhuth & Seiffert, 672 (R.F.)
Ukil, A. C., 88 (Chl.)
Underwood, F.J., 919 (Mal.)
United Fruit Company, Boston, Mass., 15 (Mal.)
United States Naval Medical Bulletin, 36 (Mal.), (101) (Pl.), 226 (Rab.)
Unna, Jr., P., 612 (Lep.)
——, with Keil, 620 (Lep.)
Unno, S., with Ogata & Nagai, 53 (Fev.)
Urbain, A., 631 (Chl.)
——, with Normet & Chaillot, 894 (Dys.)
Urdaniz, M., with Castex, Greenway, D. & Greenway, D. F. 476 (Am.)
Uriarte, L., 98 (101), 635 (Pl.)
—— & Morales Villazón, N., 635 (Pl.)
Urtubey, L., 135 (R.F.)
Ustupny, D. I., 738 (Rab.)
d'Utra, O. & Silva, 750 (K.A.)
Uvarov, B. P., 433 (B.R.)
Uyeda, O., with Torikata, 630 (Chl.)

## V

Vagliano, M., with Livierato, 452 (Fev.)
Valcke, G., 11 (Mal.)
——, with Walravens & Bequaert, 20, 931 (Mal.)
Valdés Lambea, J., (38) (Mal.)
Vallarino, J. J., 472 (Am.)
Valle, V., (951) (Mal.)
Vallée et al, 738 (Rab.)
Vallejo Najera, A., 385 (Mal.)
Valli, V., 34 (Mal.)
Valter, V., with Jonesco, 729 (Rab.)
Van den Bergh, V. H., 480 (Dys.)
Van den Branden, F., 197, 198 (S.S.), 396 (Lab.)
—— & Henry, E., (951) (Mal.)
Van den Hoven van Genderen, J., 224 (Rab.)
—— & Dik, J. H., 224 (Rab.)
Van der Hoeden, J., 62, 442 (Und.)
Van der Kodde, W. F., 820 (Z.)
Vaucel, M., with Sicé, 813 (Z.)
Vaurs, R., with Sazerac, 127 (R.F.)
Vaz, E., 486 (Dys.)
de Vecchi, B., 768 (Myc.)
de Vecchi, G., 675 (Fev.)
Vedder, A., 524 (Z.)
Vedder, E. B., 331 (Lep.)
da Veiga, O. A., 222 (Rab.)
Vellard, J., 1002 (Y.F.)
—— & Vianna, M., 1002 *ter*, 1003 (Y.F.)
Velu, H., Bigot, A. & Eyraud, R., 737 (Rab.)
——, with —— & ——, 736 (Rab.)
Venables, J. F., 467 (Am.)
Veneroni, C., 265 (Z.), 856 (Y. & S.)
Venezuela Gulf Oil Company, 588 (Misc.)
Vera, S., 454 (Fev.)
Veras, S., 835 (Fev.)
Vercellana, G., 72 (Und.)
Verge, J., 599 (Misc.)

Vergeer, T., 537 *bis*, 538 *bis* (Hel.)
Vernencar, H. P., with de Mello, 25 (Mal.)
Verslagen en Mededeelingen Betreffende de Volksgezondheid, 915 (Mal.)
de Verteuil, E., 8 (Mal.)
di Vestea, A., 225 (Rab.)
Viala, J., 222 (Rab.)
——, with Manouélian, 217, 218, 726 (Rab.)
Vialard, 472 (Am.)
Vianna, M., with Vellard, 1002 *ter*, 1003 (Y.F.)
Vidal, J., 448 (Und.)
—— & Abella, R., 447 (Und.)
Vidale, E., with Sepulcri, 949 (Mal.)
Vickers, W. J., 89 (Chl.)
Vielle, A., with Montel, 147 (Am.)
Viéru, M., with Ciuca & Ballif, 928 *bis*, 944 (Mal.)
Viev, with Audry, 624 (Lep.)
Vigne, P., 609 (Lep.)
—— & Bourret, M., 752 (K.A.)
Villaamil, R., with Schöbl, 155 (Dys.)
Villela, A. & E. L., with Jakob & Fialho, 298 (Y.F.)
Villela, G. C., with Lacorte, 299 *bis* (Y.F.)
Villela, G. G. & Teixeira, J. C., 992 (Hel.)
Vincent, G. E., 954 (B.R.)
Vincenzo, J., 985 (Hel.)
Viswalingam, A., 510 (Oph.), 959 (Pel.)
Vitzthum, H. G., 268 (Z.)
Voegtlin, C., with Hunt, McCann, Rowntree, & Eggleston, 378 (Mal.)
Voge, C. I. B., 831 (Und.)
Voizard, F. & Leroy, D., 107 (Der.)
Voizard, F. F. E. & Rault, 666 (R. F.)
Vogel, H., with Delbanco & Mühlens, 984 (Hel.)
de Vogel, W., 85 (Chl.), 254 (Z.), 616 (Lep.)
de Vogel, W. T., 182 (G.V.)
Volpino, G. & Finocchio, M., 221 (Rab.)
Voskresensky, P. W. (258) (Z.)

## W

Wada, K., 546 (Hel.)
Wade, H. W., 615 (Lep.)
Wadia, J. H., with Mackie & Goré, 497 (Sp.)
Wagner, J., 820 (Z.)
Wagner, O., 876 (Am.)
Wagner-Jauregg, J., 943 (Mal.)
Wainstein, A., 656 (R.F.)
Wainstein, A. B., with Aristowsky, 656 (R.F.)
Wakeman, A. M., 1015 (Bl.)
Walch, E. W. & Bonne-Wepster, J., 806 *ter* (Z.)
—— & Sardjito, M., 251 *bis* (Z.)
—— & Soesilo, R., 911 (Mal.)
Walker, E. L., 1040 (Lep.)
—— & Sweeney, M. A., 1032 (Lep.)
Walker, J., 29 (Mal.)
Wall, F., 348 (B.R.)
Wall, R. E., 803 (Z.)
Wallace, R. B., 374 (Mal.)
Walravens, P., with Valcke, G. & Bequaert, M., 20, 931 (Mal.)
Walter, H., with Fiessinger, 1028 (Bl.)
Warasi, W., 29 (Mal.)
Warburton, C., 267 (Z.)
Ward, C. B., with Magath, 145 *bis* (Am.)
Ward, H. B., 536 (Hel.)
Warner, W. P., with Scozzafave, 63 (Und.)
Warnowsky, I., (993) (Hel.)
Warren, S. H., with Charles, 828 (Und.)
——, with Clayton, 894 (Dys.)
Warthin, A. S., 537 (Hel.)
Watanabe, K., 983 (Hel.)
Watson, C. J., 804 (Z.)
Watson, M., 31 (Mal.)
Watson, W. H., 525 (Hel.)
Wayson, N. E. & Badger, L. F., 337 (Lep.)
Webb, U. R. & Muller, F. W., 728 (Rab.)
Webster, W. J., 811 (Z.)
Weidling, K., 243 (Z.)
Weil, A. J., with Braun, 479 (Dys.)
Weinberger, M., with da Rin, (74) (Und.)
Weir, H. C., (862) (Y. & S.)
Weiss, C., 503 (Sp.)
——, with Galbreath, 769 (Myc.)
—— & Landrón, F., 502 (Sp.)
Weiss, P., 329 (K.A.)
——, with Monje, 55 (Fev.)
Weissbrod, B., with Legezynski, 221 (Rab.)
Weitzman, I., 900 (Dys.)
Welch, T. B., 338 (Lep.), 581 (Misc.), 896 (Dys.)
Wenckebach, K. F. & Aalsmeer, W. C., 352 (Bb.)
——, with ——, 517 (B.R.)
Wenyon, C. M., 312 (K.A.)
Werne, J., with Boettiger, 986 (Hel.)
West African Medical Journal, 1015 (Bl.)
West, R., 497 (Sp.)
Westphal, F., with Cambessedès, 1024 (Bl.)
Wethmar, R., 30 (Mal.), 219 (Rab.)
Wheeler, G. A., with Goldberger, Sydenstricker, King & others, 356, 963 (Pel.)
Whitaker, E. J., 16 (Mal.)
White, C., 103 (Der.)
White, G. F., with Kirby-Smith & Dove (993) (Hel.)
Whitmore, E. R., 1011 *bis*, 1026 (Bl.)
Wichmann, F. W., 889 (Dys.)
——, with Schlossberger, 664 (R.F.)
Wiener, M., 504 (Oph.)
Wigglesworth, V. B., 808 (Z.)
Wildman, O., with Williams & Curtis, 869 (Am.)
Willey, C. H., with Stunkard, 974 (Hel.)
Williams, D. P., with Strickland, Chowdhury, Dodds-Price, Forsyth, Smith & Murphy, 4 (Mal.)
Williams, H. U., with Rice, J. P. & Lacayo, J. R., 284 (Hist.)
Williams, L., with Tegoni & Frugiuele, (38) (Mal.)
Williams, L. H., 860 (Y. & S.)
——, Wildman, O. & Curtis, L. F., 869 (Am.)
Williamson, C. S., Kaplan, B. & Geiger, J. C., 870 (Am.)
Williamson, K. B., 368 (Mal.)
Willmott, C. B., 182 (G.V.)
Willoughby, H., 467 (Am.)
——, with Cooke, 1021 (Bl.)
——, with Manson-Bahr, 825 (Und.), 145, 873 (Am.)
Wilmoth, C. L., 184 (C.Bu.)
Wilshusen, H. F., with Hillsman & Zimmerman, 184 (C.Bu.)
Wilson, C., (600) (Misc.)

Wilson, J. H., with Page & Turner, 592 (Misc.)
Wilson, P. W., (38), 922 (Mal.)
Wilson, R. M., 624, 1036 (Lep.)
—— & Gilmer, W. P., 1041 (Lep.)
Wilson, R. P., 505 *bis*, 507, 509 (Oph.)
Winckel, C., 85 (Chl.)
Wirssaladse, S., 934, (951) (Mal.)
Wischnevsky, N. C., 924 (Mal.)
Wise, F., 341 (Lep.)
Witenberg, G., 974 (Hel.)
Wofcy, I., 122 (R.F.)
Wollman, E., Anderson, C. & Colas-Belcour, 242 (Z.)
Wolski, M. E. & Schewelewa, E. M., 927, 938 (Mal.)
Wolter, F., 627 (Chl.)
World's Health, 610 (Lep.)
Wright, F. C., 609 (Lep.)
Wright-Smith, R. J., 1030 (Bl.)
——, with Fairley, 979 (Hel.)
Wu Lien Teh, 98, 100 (Pl.), 518 (B.R.)
Wupperman, R. C., 588 (Misc.)
Wyckoff, H. A., with Bloomfield, 496 (Sp.)

Y

Yagi, T., 463 (Fev.)
Yamada, S., 543 (Hel.)
Yamaguchi, T., 353 (Bb.)
Yamanaka, S., 23 (Mal.)
Yamashita, H., (489) (Dys.)
Yampolskaya, F. S., with Bogoyavlensky, (37) (Mal.)
Yang, C. S. & Berglund, H., 937 (Mal.)
Yeager, C. H., 1045 (Misc.)
Yoannovitch, G., 140 (Am.), 252 (Z.)
Yoel, M., with Papadopoulo & Hadjigeorges, 835 (Fev.)
Yokogawa, S., 992 (Hel.)
Yoshitake, S., 873, 885 (Am.)
Young, C. W. & Hertig, M., 741 (K.A.)
Young, T. C. M. & Baily, J. D., 362 (Mal.)
—— & Majid, A., (258) (Z.), (951) (Mal.)
Young, W. A., 389 (Lab.), 547 (Hel.)
Yuan-Po, L., 798 (Z.)

Z

Zahorsky, J., 164 (Dys.)
Zaikine, N. A., (258) (Z.)
Zallocco, A., 12, 925, 926 *bis* (Mal.)
——, with Genoese, 370 (Mal.)
Zanzucchi, A., 832 (Und.)
Zavagli, V., 710 (S.S.)
Zdrodowski, P., 90 (Chl.), (993) (Hel.)
Zeiss, H., (101) (Pl.)
—— & Popow, P., 490 (Sp.)
Zia, S. H. & Faust, E. C., 29 (Mal.)
Ziemann, H., 946, (951) (Mal.)
Zimmerman, C. B., with Wilshusen & Hillsman, 184 (C.Bu.)
Zimmermann, E., 672 (R.F.)
Zlatogoroff, S. F. & Moghilevskaïa, B., 99 (Pl.)
Zlatogoroff, S. I. & Moghilewskaja, B. I., 640 (Pl.)
Zlatogorov, S. & Mogilevskaya, B., 640 (Pl.)
Zschucke, J. (903) (Dys.)
Zubriczky, A., 286 (Hist.)
Zuelzer, M., 426 (B.R.)
——, with Sardjito, 670 (R.F.)

# INDEX OF SUBJECTS.*

Compiled by MISS M. H. JAMES.


**Abortus Fever,** *see under* **UNDULANT FEVERS**

**Ainhum** in Negress: Alabama, **690**

**AMOEBIASIS** (AMOEBIC DYSENTERY, LIVER ABSCESS, &c., *see also* **DYSENTERY**), 140–65, 465–89, 869–903
- Abscess, Pulmonary, Non-Amoebic, Cured by Emetine, **880** *bis*
- Amoebae and Entamoebae associated with Haematophagous Dysenteric, Test of, for Iron, **886**
  - Pathogenic Culture Media for, for Routine Cultures of Faeces for, **885**
  - Species referred to
    - *coli*, *histolytica* and *Dientamoeba fragilis*, Transmission of, to Rats, **884.**
    - *dysenteriae*, *see histolytica, infra*
    - *histolytica*
      - Carriers of
        - Disease-Incidence in Relation to, **144**
        - Symptomatology; Diagnosis: Treatment, **143**
      - Cultivation of **475, 885**
        - Difficulty in, **150**
      - Culture Media for
        - New, **474**
        - Various, **474**
      - Cultures of
        - Action in, of Emetine, **469**
        - Made with Rice Starch, Pathogenic Power of, **886**
      - Cysts of, Effects on, of Certain Chemicals, **150**
      - Infection with, **140** *bis*
        - Complement Fixation in, **150**
        - in Egyptian Children, 162
        - in Holland, **870**
        - Intestinal, of Man, **140** *bis*
        - in Kittens, Blood Cells of, Changes in, **883**
      - in Pleural Effusion, **880**
      - Presence of, with *Trichomonas hominis*, Dysenteric Symptoms with, **475**
      - Protozoophagy by, **152**
      - *in Vitro* Effects on, of Radium with and without Metallic Sensitizers, **878**
    - in Western Colombia, 586
- AMOEBIASIS
  - CEREBRAL, in an Annamite, **147**
  - DYSENTERIC, *see* AMOEBIC DYSENTERY, *infra*
  - EXTRA-INTESTINAL: Diagnosis, **465**
  - HEPATIC, Siam, **466**
  - PULMONARY, 465
    - Treatment; Pneumothorax and Emetine, **470**

**Amoebiasis**—*cont.*
- AMOEBIASIS—*cont.*
  - URINARY, 465
    - Alleged, with Haematuria, **881, 882**
  - AMOEBIC ABSCESS, *see also* LIVER ABSCESS, *infra*
    - Cavities of, Irrigation of, by Carrel-Dakin Method, **879**
  - AMOEBIC BRONCHITIS, with *Treponema bronchialis*, **476**
  - AMOEBIC CHOLECYSTITIS, Reality of, **468**
  - AMOEBIC DYSENTERY
    - Acute, **874**
    - Appendicular Syndrome in, **147** *bis*
    - Carriers of, Symptomatology, Diagnosis, and Treatment, **143**
    - Chronic
      - Cure by Montel's Method, **882**
      - and Latent, Surgical Complications of, **875**
    - Complications of, **468, 469, 875**
    - Deaths after Emetine Injections, **882, 883**
    - Diagnosis
      - Laboratory Methods, **145** *bis*
      - Leucocyte Count in, Value of, **145**
    - Experimental, in
      - Kittens, 151
      - Rats, with *E. histolytica*, *E. coli* and *Dientamoeba fragilis*, **884**
    - Extent of Infection of, Estimation of, Value in, of Roentgen Rays, **472**
    - Extra-Intestinal, Diagnosis of, **465**
    - Incidence
      - Age: Infants and Children, **163, 876, 883**
      - Animal
        - in Kittens
          - Blood Cell Changes in, **883**
          - Pathogenesis of, **473**
      - Geographical
        - America, Central and South, 140 *bis*
        - Belgian Congo: Kasai Mines, 410
        - Brazil, **163**
        - Britain, **467** *bis*
        - China: Yunnan, 416
        - Egypt, 486
        - France: Asylum Epidemic, **475**
        - Haiti, **869**
        - Holland, **870**
        - India: Kohat, **896**
        - Italy: Bologna, **467**
        - Morocco, chiefly in Fez, **140**
        - Manchuria: Dairen, 162
        - Palestine, **876**
          - Investigations into, **899**
          - Tiberias, 883
        - Porto Rico, **466**
        - Senegal: Dakar, 468
        - Serbia, Southern, **140**
        - Siam, **466**


* The numbers in heavy type refer to the titles of papers summarized.

**Amoebiasis**—*cont.*
AMOEBIC DYSENTERY—*cont.*
Incidence—*cont.*
Geographical—*cont.*
U.S.A.
Chicago, **870**
New Orleans, 470
Venezuela, 588
Leucocyte Count in, as Aid to Diagnosis, **145**
Pericarditis during, **468**
Pleuritic Effusion with Presence of *E. histolytica*, **880**
Sigmoidoscopic Appearances in, 159–60
Treatment, **148**
in Infants and Children, **876**
of Chronic Cases, **471**
Mayo Clinic Results, **148, 149**
Specific, **470**
Various Methods
Emetine, **877**
Poisoning Cases; Deaths after Injections, **882, 883**
Emetine Bismuth Iodide and Emetine Periodide, Possible Cumulative Effect of, **470**
Montel's Method, **882**
Paroxyl, 869
Rivanol, **876, 877, 900**
Spirocid, **901**
Yatren, 148, 149, **149** *bis*, **472** *bis*, **475**, 577 *bis*, **877, 878** *bis*, **900**
Uro-genital Symptoms Probably Amoebic: Cure by Emetine, **471**
AMOEBIC HEPATITIS
Chronic, with Periodical Relapses, **872**
Incidence
India, 469
Morocco, **468**
Siam, **466**
Periostitis of Tibia following, **469**
Treatment
Whipple's Method as Adjuvant in, **472**
Yatren, **878**
AMOEBIC ULCER of Abdominal Wall, following Appendectomy with Drainage, **874**
Appendicitis of Amoebic Origin, Symptoms of, **476, 875**
Eleven Papers on, Summarized, **871**
Titles of Unnoticed Papers, 165, 489, 902–3
LIVER ABSCESS
Anaemic Form of, **873**
Cases of, **146** *bis*, **873** *bis*
One Acquired in England, **146**
Cured by Spontaneous Emesis, **873**
Incidence
England, **146**
Formosa, 873
Morocco, **468**
Siam, 466
Leucocyte Count in, **873**
Masked by Pleural Symptoms, **469**
Rupture of, into Abdominal Cavity and Inferior Vena Cava, **469**

**BERIBERI,** 349–55, 964–8
Book on, Reviewed, **517**
Cardiac Affections in
Heart Failure, **352**
Nature of, **352**

**Beriberi**—*cont.*
Cardio-vascular Beriberic States, Radioscopy in, **964**
*Columbarum*, Vitamin B, Deficiency in, **966**
Diet in relation to (*see also* Vitamin, *infra*), 965
Epidemic Dropsy Form, **353**
Outbreaks of: India, **354, 355, 967** *bis*, **968** *bis*
Etiological Theories
Oedema Theory, **964**
Studies in, **965** *bis*
Gall-Bladder in, Gross and Histological Changes in, **351**
Human, Does Avitaminosis Explain? **350**
and Human B Avitaminosis, Fluid Exchange in, **353**
Incidence
Geographical
India
British
Allahabad, Epidemic (1927), **354**
Birbhum, Recent Outbreak, **667** *bis*
Burma: Rangoon, **968**
Surada, Epidemic (1924–5), **355**
Portuguese: Goa, **968**
Malaya: Singapore, 351
Persian Gulf, on Ship, **964**
Philippine Islands
Report of Committee on, **349**
Syria, 350–1
Race, 350–1, 964
POLYNEURITIS AVIUM
Vitamin B Deficiency in, in Pigeons, and occurrence in of, Refection, **966**
Relation of, to Polyneuritis from other Causes, **350**
Rice in relation to, **964**
Title of Unnoticed Paper, 355
Vitamin B
Deficiency of, in Pigeons, *see* POLYNEURITIS AVIUM, *supra*
Physiological Action of, **964**

**BLACKWATER FEVER,** 1009–30
Blood in, **1026**
Bilirubin Estimation in, **1028**
Cases Studied at Liverpool, **1016**
Distribution of, in India, Burma, the Far East and Africa, **1011** *ter*
Epidemiology (*see also* Incidence, *infra*), **909**
Etiological Hypotheses, **1009**
Incidence
Age: Children, **1025** *bis*
Geographical
Africa, **1011**
Cuba, 1011, **1013,** 1026
Far East, **1011**
India, **1011**
Bengal Duars, **909, 1027**
Burma, **1011**
Italy, 1025
Jamaica, 1011
Kenya, 907
Nigeria, **1015**
Palestine, **1017**
Panama, 1011
Salonica, **1020**

**Blackwater Fever**—*cont.*
Incidence—*cont.*
Geographical—*cont.*
Siam: Epidemiology of, **1017**
Uganda (1927), **1014**
U.S.A.: Southern States, 919
Race, 1011
Kidney Changes in, **1019** *bis*
Malarial Haemoglobinuria, **1011** *bis*
and Other Haemoglobinurias, **1009**
Pathogenesis of, **1018, 1020**
Quinine in, *see under* Treatment, *infra*
and Quinine, Statistical Note, **1029**
Quinine, Haemolytic Action of, **1028**
Quinine as Provocative of, **1029, 1030**
Quinine-Haemoglobinuria in Malaria, Haemolysin of, **1029**
Symptoms of, Interpretation of, **1018**
Treatment, **1020** *bis*
Suggestion for, **1018**
Various Methods
Blood Transfusion, **1023, 1024**
Recovery after, **1030**
Plasmoquine, **1030**
Quinine, **1024**
Intolerance thereof, in African Child, **1025**
Quinine Collobiase, **1030**
Serums and Alkalies, **1022**
Sod. Bicarb. Solution, **1021, 1022** *bis*
Stovarsol, **1026**
Urine Retention in, **1026**

**CHOLERA,** 83–90, 626–32
Asiatic, **86**
Bacteriology
*Vibrio cholerae*
Action on, of Cholera Convalescent Serum, **88**
Haemolysin, The, of, **631**
Metabolism of, in Aerobic and Anaerobic Culture, **628**
Motility of, Oxygen and, **87**
Normal and Sensitized, The Electric Charge of, and of their Agglutinins, **88**
Passage by, of Intact Skin, **87**
Permanent Hanging-Drop Phenomenon, **87**
Vibrios allied to, Epidemic Diarrhoea due to: Gabon, **86**
Vibrios Resembling, from Human Beings and from Water in Manila, Serological Relationships of, **89**
Bacteriophage of
Presence, Virulence, and Use of, as Preventive or Treatment, 86
Work on, Report on, **90**
Carriers of, in S.S. *Hawaii Maru*, **85**
Cholera Antigen
Action of, Heated and Unheated, **629, 630** *bis*
Impedin Phenomenon, and Spontaneous Phagocytosis, 629, **630**
Production by, of Agglutinins and Bacteriolysins, **629**
Toxicity and Immunizing Power of, **630**

**Cholera**—*cont.*
Cholera Convalescent Serum, Action of, on Comma Vibrios, **88**
Cholera Research, Foundations of: Broad Street Pump. The Pettenkofer Memorial Essay, **627**
Clinical Signs, Certain, in, Incidence and Significance of, **626**
Diarrhoea, Epidemic, due to Vibrios: Gabon, **86**
Experimental, by the Intraspinal Route, **631**
Incidence
China, Problems of, **84**
Dutch East Indies
Batavia: 1927 Epidemic, **85** *bis*, **627, 632** *bis*
Federated Malay States, **626**
India
Epidemics of, Forecasting and Control of, **83**
19th Century and up to 1922, **83**
Asansol Mining Settlement, **626**
Bengal, 1927–8, and 1927, **83, 89**
Calcutta, 627
Bombay Presidency, 1927 Epidemic, **83**
Madras, 1927, **89**
Indo-China, **84**
Japan, Problems of, **84**
Pathogeny, Experiments on, **90**
Possible Cases of, Value [for Identifying] of Investigating Diarrhoeal Diseases, **626**
Prophylaxis
Anticholera Vaccine for, Large-scale Manufacture of: Saigon, **632**
Bacteriophage, 86
Control Measures in
Federated Malay States, **626**
Rural Areas, especially Rubber Estates, **89**
Vaccination
in British India, 1927, **89**
Rapid, Instructions for, **85**
Titles of Unnoticed Papers, 90, 632
Toxin of, Enterotropism a Parenteral Action of, **628**
Treatment
in Federated Malay States, **626**
Present-Day, **627**
Various Methods
Bacteriophage, 86
Cholera Convalescent Serum, **88**
Kaolin, **87**

**CLIMATIC BUBO,** 184, 862–3
Differential Diagnosis, **598**
Incidence
Brazil, 184
Nigeria, **174**
Serbia: Belgrade, 862
Spain: Madrid, **863**
U.S.A., 184 *bis*
Inoculability of, Experimental, **863**
with Large Iliac Glands, **862**
Lymphogranuloma Inguinale, **863**
with Possible Reinfection or Superinfection, **862**

**Climatic Bubo**—*cont.*
Reports of Cases, **184** *bis*
Treatment
Methylene-blue, **184**
Protein-shock, 174

**DENGUE, AND DENGUE AND PAPPATACI FEVERS** (*see also* **UNCLASSED FEVERS**), 39–46, 450–7, 832–42
DENGUE
*Aëdes aegypti*
Infected with Virus of, Rickettsia in, **43, 837**
Duration of Infection as Influenced by Cold Season, **838**
Transmitter of, **43,** 833, 834, 835, 840
Blood in, **837**
Colorado, Form of, so-called, **41**
Complications of
Fatal, **840**
Surgical, **835**
Dengue, **44**
Dengue: Present State of Knowledge on, **457**
Diagnosis, Early, of, Test in, of the State of the Peripheral Vessels, **453**
Effects of, on Kidneys, **836**
Epidemic of, Clinical Description of, **455**
Etiology, Researches on, **41, 44** *bis*
Experimental Work on, **42**
Identity of, Probable, **843**
Immunity to, Does an Attack confer? **839,** 840
Incidence
Age: Children, **454, 835**
Geographical
Australia, **46** *ter*
New South Wales, 1925–6 Epidemic, **40**
China, North, in Winter, **455**
Egypt, Epidemics (1927 and 1928), **39,** 840
Far East, **455**
French Indo-China, Saigon, 455
Greece, Epidemic of 1927–8, **40, 450** *sqq.*, **833** *bis*, **834, 835** *ter*, **836,** 837 *bis*, 838, 839, 840, 842
Clinical Study of, **451**
New Caledonia, 456
Philippines; Study of 100 Cases, **841**
Manila, 455
Rhodes, Island of, Epidemic (1928), **842**
Senegal
Dakar, Naval Station, 1927 Epidemic, **39**
Saint-Louis: Epidemic (1926), **841**
Soudan, French, **39**
Spain
Andalusia, Epidemic, **41** *bis*
Palestine
Tiberias, Epidemic, **840**
Shipboard. 455
Infection with, Inapparent, in Man and Guineapig, **456**

**Dengue, and Dengue and Pappataci Fevers**—*cont.*
DENGUE—*cont.*
Jaundice Complicating: Relation to Yellow Fever, **456**
Malaria in relation to **842**
Monkey-susceptibility to, Experiments on, **838**
and Red Fever of the Congo, **46**
Special Clinical Types of, and their Treatment, **452**
Transmission by *Aëdes aegypti*, **43,** 833, 834, 835, 840
Transmission Experiments on, in Greece, **453**
Tuberculosis and, **836, 837**
Vagotonic Syndrome in, **453**
Virus of, Action on, of
Anti-Yellow Fever and Hog Cholera Sera, **839**
Serum and Whole Blood of Convalescents, **840**
Duration of, in *Stegomyia*: Influence of Cold Season, **838**
Rickettsia Forms and Inclusions in *Stegomyia* Infected with, **837**
PAPPATACI FEVER
Etiology, **44** *bis*
Incidence
Baluchistan, **47**
French Equatorial Africa
Abéché, **457**
Tiflis, Epidemic, 45
Titles of Unnoticed Papers, 46, 842

**DERMATOLOGY, TROPICAL** (*see also* **MYCOLOGY**), 102–12, 677–90
Allergy in, Clinical Aspects of, **679**
Idiosyncrasy and, Rôle of, **677**
Dermatological Experience in India, **677**
DISEASES
Actinomycosis with Yellow Granules, **683**
Ainhum, *see that head*
Aroeira, of Southern Brazil, **108**
Carate (Pinta), in Colombia, **105**
Wassermann, Kahn, and Meinicke (M.T.R.) Reactions in, **105**
Chromoblastomycosis: Cuba, **104** *bis*
Experimental: Brazil, **685**
Cooly Itch, due to *Trichophyton violaceum* var. *indicum*, **682**
Creeping Eruption (*see also under* **HELMINTHIASIS**)
Etiology, **688**
Incidence
Greece, **687**
Lagos, **688**
Natal, called Sand-Worm Disease, **687** *bis*
Dermatitis
Papular, Epidemic in Belgian Congo, **688**
Verrucosa, *see* Chromoblastomycosis
Dermatomycoses
Brazilian, **102**
Due to *Penicillium crustaceum*; Tripoli, **684**
General Remarks on, **679**
Haematogenous Production of, **679**
Tropical, **102**

**Dermatology, Tropical**—*cont.*
DISEASES—*cont.*
Dermatosis Papulosa Nigra, **689**
Dermoepidermosis, Epidemic, Papular; Lower Congo, **110**
Elephantiasis Neuromatosa, in a Tamil, **690**
Fungus Infections of Hands and Feet, U.S.A:, **680** *bis*, **681**
Treatment by a Fuchsin Paint, **681**
Lupus erythematosus in Black Races, **111** *bis*
Lymphadenitis Filarial, *see under* **HELMINTHIASIS, Filariasis**
Mycotic Inguinal, associated with Pedal Dermatitis, **103**
Madura Foot, *see* Mycetoma pedis, *infra*
Met with in Western Colombia, 586
Mongolian Blue Spots among Filipinos, **111**
Mycetoma Infection: an Appeal for Material, **106**
Black Grain, due to hitherto Undescribed Torula, **106**
of Hand and Foot, **108**
*pedis*: Tripolitania, **683**
Cure by
Chemotherapy, Apparent, **108**
Lugol, **107**
Red Grain, Nigeria, **107**
White Grain: Paraguay, **683**
Naga Sore: Assam; Etiology: Prevention, **686** *bis*
Onychia, associated with *Acrotheca pedrosoi*: Brazil, **684**
Paaj, a Form of Dermatitis Venenata, **686**
Provoked by the Red Quebracho, **109**
Piedra, Paraguayan, **683**
Cyst-Development in, **106**
Neo-Endothrix Fungus in, **684**
Pityriasis versicolor, Tropical, Facial, followed by Achromia, **103**
Plant-caused, 108 *bis*, **109**
Prickly Heat, and the Seborrhoides, **108**
Pseudomycosis due to a Coccus, **684**
Ringworm
Algeria; Southern Oran, **681**
of Feet, **102, 103** *bis*
of Scalp: Algeria, **682**
Scabies
History of, **289**
Treatment, **112**
Skin, in Siam, **102**
Sporotrichosis, Familial: Brazil, **685**
Tinea flava (Castellani), **104**
*Trichomycosis palmelina*, Exanthema of the Axilla due to, **108**
Trichophytosis, Experimental, of Algerian Monkey, **103**
Ulcers: Treatment; Pix Liquida, **595**
Ulcus Molle, Buboes, Lymphogranuloma Inguinale, and Climatic Bubo, Differential Diagnosis of, **598**
Ulcus Molle Serpiginosum, Case of, **598**
Ulcus tropicum
and Dietary, **109, 110**
Incidence
Belgian Congo: Kasai, 411
Nigeria, Northern, **109**

**Dermatology, Tropical**—*cont.*
DISEASES—*cont.*
Ulcus tropicum—*cont.*
Treatment, **685, 686**
Various Methods
Dried Human Serum, **595**
Desitin, **685**
Idiosyncrasy and Allergy in, Rôle of, **677**
Titles of Unnoticed Papers, 112, 690

**DYSENTERY,** BACILLARY, CILIATE, FLAGELLATE, MIXED AND UNCLASSED (*see also* **AMOEBIASIS**), 152–65, 476–86, 886–903
Acute, Castor Oil Treatment for, **896**
Bacteriology
*Bacillus(i)* (Bact.) associated with *ceylonense*, **895**
*dysenteriae*
Agglutinins to, in the Apparently Healthy, **890**
and Bacilli of the Typhoid Group, Bacteriological Exam. for, of Faeces, **477**
Biochemistry of, **480**
Exotoxin of, Physiological and Immunological Studies of, **485**
California Strain, **479**
Flexner, Infection in Two-day-old Child, **479**
Serum of, Agglutinins and Agglutinogen of, Action on, of Heat, **888** *bis*
Shiga, and Y, in Egyptian Children, 162
Forms Isolated in Roumania, **154**
Hiss
Mutation of, with
Culture in Homologous Antiserum, **892**
Subjection to a Lytic Principle, **892**
Kruse (Type E), **478**
Kruse-Sonne, **479**
Mannite Fermenting, Toxicity of, Nature of, **157**
Shiga, Constituents of, Immunological Studies of, **481**
Sonne, Dysentery in New York Attributed to, **895**
Fulminating Case due to, in Child, **154**
Investigation of, **153**
Sonne Type, Infection with: Lahore District, **478**
Type E (Kruse), **478**
Types of: Kyoto, **477**
Metadysentery and *B. coli anaerogenes*, **477**
Metadysentery Group, **155**
Paradysentery, Case due to, **895**
Unusual, **894** *bis*
Classification of, **155**
Complications of, in Child, **895**
Diagnosis, **886**
Bacteriological, **155**
Agglutination Reaction in, Limitations of, **155**

**Dysentery**—*cont.*
Dysentery Bacteriophage, Preparation of, for Therapeutic Use, **480**
Dysentery Vaccine(s)
Shiga-Kruse Anavaccine, Properties of, **891**
Sterilization of, by Heat and by Yatren, **891**
E. Dysentery, Clinical and Bacteriological Characters of, **894**
Incidence
Age, Children and Infants, **478, 479, 895**
Geographical
Annam, 894
Brazil
Joinville, 896
Rio Grande do Sul, **893**
Egypt, 486
England, 894
Newcastle: Sporadic Cases, **153**
India
Khasi Village, **901**
Lahore District, **478, 893**
Western Command, **485**
Japan: Epidemic, **163**
Kyoto, **477**
Kenya, 896
Madagascar, Epidemic, 1928, **479**
Manchuria
in Children: Bacillus Types: Blood Findings, **478**
Dairen, 162
South, 487
Palestine, Investigation of, **899**
Philippine Islands, **152**
Trinidad, 896
Uganda, 896
U.S.A.: New York, **895**
Race
Relative Mortality in Chinese and Japanese, 162
Joint Affections, Chronic, after, **902**
Metadysentery and the Metadysentery Bacilli, **155, 477**
Organisms of, Biochemistry of, **480**
Post-dysenteric Spastic Constipation, **488**
Prophylaxis
Antidiphtheritic and Antidysenteric Sera, Action of, Some Conditions of, **481**
Anti-Dysentery Serum(a) for
Agglutinating, from Horse, Use of, in Practice, **156**
Agglutinin and Antitoxin Content of, Comparison of, **484**
Specific, Procural of: Use of, in Classification, **887**
Standardization of, **482, 890**
Testing of, Statistical Note on, **482**
Titration of, **156**
Accuracy of, Effect on, of Increased Dose of Toxin, **482**
Immunization against, Oral, **485** *bis*
Vaccination, Antidysenteric Vaccine for, Preparation of, **486**
Sigmoidoscopic Appearances in, 160–1
Spread of, Control of, in a Lunatic Asylum, **476**
by Water (probably), **901**

**Dysentery**—*cont.*
Titles of Unnoticed Papers, 165
Toxin(s) of
Abiuretic, Purified, **483**
Influence of, on Rabbit's Gut, Relation between Age and, **484, 485** *bis*
Nature and Purification of, **483**
Neutralization of, by means of Cholesterin, **484**
Production of, in Synthetic Media, **889**
Shiga, Mice-Susceptibility to, Seasonal Variation in, **889**
Ultrafiltration of, **157**
Treatment, Various Methods
Bacteriophage, **901**
Castor Oil, in Acute Cases, **896**
Rivanol, **900**
Vaccination
Oral and Oral and Subcutaneous by Living Bacilli, **158**
Yatren, **900**
Enemas of: Rapid Cure, **896**
CILIATE-BALANTIDIASIS
*Balantidium coli* of, Pathological Significance of, **488**
Incidence
Dutch East Indies, **902**
Egypt, 486
Siam: Chiengmai, 164
Treatment
Ol. Chenop., **164**
Santonin, **902**
FLAGELLATE
GIARDIASIS=LAMBLIASIS
Diet in relation to, **163**
Incidence
Age, **164**
Geographical: Egypt, 486
*Lamblia intestinalis* in Case of Cholelithiasis, **488**
MIXED AND UNCLASSED, 159–65, 486–9, 896–903
Acute Bacillary Intestinal Infections in Egypt, Investigations of, **897**
Book on, Reviewed, **77**
Colitis, Ulcerative, **487**
Diarrhoea and Dysentery of Children: Egypt, Micro-organisms of, **161**
Diarrhoea and Enteritis in Porto Rico; Relation of, to Water Supplies, **162**
Dysenteries of Tropical Origin, Sigmoidoscopic Appearance of, **159**
Enterocolitis, Dysenteriform, Severe, in Infants: Santiago del Estero, **487**
Incidence
Egypt, **897**
Etiology, **486**
Espiritu Santo, 416
India, Kohat District, **896**
Kenya, 407
Manchuria
Dairen: Forms seen, **162**
South, **487**
Nodular, So-called, **899**

**Dysentery**—*cont.*
Treatment by
Rivanol, **900**
Yatren 105, 577 *bis*, **900**
Triple Intestinal Infection in, **488**
Urine of, Hydrogen-Ion Concentration of, **489**

**ENTERIC FEVERS IN THE TROPICS**
TYPHOID
Malaria Coincident with: Porto Rico, **925**
Prophylaxis
Vaccination, Oral, **597**
TYPHOID AND PARATYPHOID, in Egypt, 897

**Framboesia,** *see* YAWS *under* **YAWS AND SYPHILIS**

**Gangosa,** *see under* **YAWS AND SYPHILIS**

**Goundou**
Cases resembling in
Man, **174, 856**
Pony, **857**
in Gorilla, **857**
Incidence
Nigeria, **174**
in Native, 173
Panama, in Jamaican Native, **174**
and Yaws, **173**

**GRANULOMA VENEREUM,** 181–3, 863–6
Diagnosis, **864**
Donovan Body of, 181, **181**
Culture of, **866** *bis*
Etiology, **181**
Granuloma Venereum, **864**
Granulomata, Perianal, **865**
Incidence
British Guiana, **864**
Dutch New Guinea
Campaign against, **182, 183, 863**
Norway, in Seaman, **183**
Paraguay, 865
U.S.A., 181 *bis*, 182
Venezuela, 864
Prophylaxis, in Dutch New Guinea, **182, 183, 863**
Reports of Cases, **181, 182, 183**
Spirochaetes and Treponemata from Case of, **667**
Treatment
Antimony Pot. Tart. in Glucose, and by Protein Shock, **865**
Stibenyl Heyden, **864**
Stibosan, **866**
Ulcerating, in Ribbon-like Form, **866**
and Sclerosing, **182**

**HELMINTHIASIS,** 525–58, 969–93
Books on, Reviewed, **432, 512, 513, 605, 867**
DISEASES, and Parasites associated with them
ANGUILLULOSIS, and Syndroms of "Idiopathic Hypereosinophilia," **988**

**Helminthiasis**—*cont.*
DISEASES—*cont.*
ANKYLOSTOMIASIS
Anaemia of
Blood Study in, **992**
Erythrocytes in Regeneration Appearances of, **555**
Book on, Reviewed, **605**
Historical, **555**
Incidence
Argentina, 990
Belgian Congo, Kasai Mines, 411
Ceylon, **552** *bis*
Fiji, 555
Georgia: Abkhasia, **993**
India, 991
Italy: Modena, **993**
Nyasaland, North, **525**
Porto Rico, **552**
U.S.A.
Georgia, in School Children, Decatur County, **551**
Parasites associated with
Ancylostoma
Action on, of Chrysemine, **556**
and Allied Parasites, Changes of Blood Features in Parasitisms of, **556**
Infection by, Differentiation of, from *Necator* Infection by Relative Amount of Eosinophilia, **556**
Larvae
Development of, in Uterus of Dead Female, **991**
Effect on, of Light and Drying, **554**
Penetration by, of Shoe-material, **553**
Loss and Acquisition of, Rates of, **991**
Ova
Development of, in Soil and Water and Longevity of Larvae in them, **556**
Faeces containing, Preservation of, Simple Method for, **553**
in Stools, Counting Methods of Caldwell and Stoll Compared, **553**
*braziliense* (Cat and Dog Hookworm), Experimental Creeping Eruption from **555**
in Formosan Aborigines and their Cats, **992**
*caninum* and *A. ceylonicum*, Third Stage Larvae of, **554**
*caninum*
Strain of Adapted to Cats, **992**
*duodenale*, and *Ascaris lumbricoides*, Ova-Parasite Ratio for, **553**
Natural Infectious, Road of, **556**
Prophylaxis
in Georgia, **993**
Periodic Mass Treatment as Temporary Control Measure; Ceylon, **552** *bis*
Pit Latrines in, 552

**Helminthiasis**—*cont.*
DISEASES—*cont.*
ANKYLOSTOMIASIS—*cont.*
Treatment, Mass, Historical Reference to, **555**
APPENDICITIS, *Oxyuris* present, **990**
ASCARIASIS
Abscesses, Multiple, in Liver, Secondary to, **550**
Anaemia, Extreme, with, **550**
Ascarids associated with
Body Fluid of, Haemoglobin in, **551**
Embryos of, in Mammary Glands, **551**
in Infants ; Ankole, 525
Toxin of, Action of, Experimental Studies on, **550**
*lumbricoides*
in Cavity of Human Heart, **986**
Extracts of, Cutaneous and Precipitin Tests with, **549** *bis*
Infection through the Skin, Possibility of, **548**
Occlusion by, of Larynx : Death, **987**
and *suum*, Ova of, Longevity of, **548**
Toxins of, Changes in the Toxicity of Smooth Muscle Produced by, **549**
Complications in, **550** *quat.*
Eosinophilia in, Probable Origin of, **986**
Incidence
Malaya, 550 *ter.*
Switzerland : Exam. for Ova, **986**
U.S.A., 986
Virginia, **985**
Intestinal Obstruction due to, **550**
Nephritis of : Fatal Case, **987**
with Severe Clinical Symptoms, **987**
CREEPING ERUPTION or Larva Migrans (*q.v., under* **Dermatology**) in North Queensland : Note on *Gnathostoma spinigerum*, **989**
ECHINOCOCCOSIS=HYDATID DISEASE
Books on, Reviewed, **605, 691**
*Echinococcus*(1) of
Antigens of, **541**
Scolices, Extracts of, Anaphylactic Studies with, **978**
Hydatid Sand, Action on, of Ultraviolet Rays, *in vitro*, **979**
Incidence
Australia, 978, 979
New Zealand : a Clinical Problem, **978**
Intradermal Test in, **977**
Secondary, **980**
Serological Diagnosis of, in Man, **541**
in Sheep, Oxen, and Pigs ; Daughter Cyst Formation, **979**
FILARIASIS
Book on, Reviewed, **513**
with Chylocele and Periorchitis, **984**
Chyluria
due to *F. bancrofti*, in
Canada, **547**
Italy, **985**
Occurring after Confinement, **545**

**Helminthiasis**—*cont.*
DISEASES—*cont.*
FILARIASIS—*cont.*
Diagnosis, Radioscopical, **984**
Dog-Filariasis, Treatment with Neostibinal, **546**
Extra-Peritoneal Abscesses attributed to, **547**
Filaria and Microfilaria associated with in Hydrocele Fluid, **546**
*bancrofti*
Chyluria due to
Canada, **547**
Italy, 985
Infection, Elephantoid, Hungary, **985**
Periodicity of, Mechanism of, **981**
Japanese Mosquitoes Suitable as Hosts for, **543**
Re-Description of, **545**
*medinensis, see* GUINEA-WORM INFESTATION *infra*
Filarial Lymphangitis, **983**
of Spermatic Cord, **983**
Incidence
Dutch East Indies, **546**
India, British, **544,** 545
Cerebral Symptoms Present, **547**
Japan : Abnormal Case, **547**
Korea, **547**
Liberia, 984
Mauritius : Port Louis, **543**
Porto Rico, **544, 545, 983**
Treatment, Studies in, **984**
by Carbon Tetrachloride, **970**
GUINEA-WORM INFESTATION=DRACONTIASIS or DRACUNCULOSIS
Incidence
Dutch East Indies, in an Arab, **547**
France ; Embryos in Abscess, **548**
LIVER FLUKE INFESTATION, **529, 530, 971, 973** *quat.*
LYMPHANGITIS with and without Filariasis : Porto Rico, **983**
OXYURIASIS
*Oxyuris* associated with
Present in Appendicitis, **990**
Two Unusual Seats of Infection in Man, **557**
*incognita*, Three Cases of Infestation by, in Man, **556**
PARASITIC INFECTIONS Unspecified, in Japan, Recent Studies on, **527**
SCHISTOSOMIASIS, or BILHARZIASIS
Chronic ; Pathology and Treatment, **971**
Incidence, all Forms
Belgian Congo, *S. mansoni* Infection, **534**
Egypt, **971** *quat.*
Appendicitis due to, 973 *bis*
Japan
Katayama Disease, 972
History of, **535**
Palestine, 971
Porto Rico, 534 *bis*, 535
South Africa, **532**
West Indies : St. Kitts, in Monkeys, 533

**Helminthiasis**—*cont.*
DISEASES—*cont.*
SCHISTOSOMIASIS—*cont.*
Nile in Egypt, as Source of Infection, **971** *bis*
Prophylaxis
Birds as Protective, **532**
Japanese, **535**
Rectal or Intestinal, Precipitin Test in, **534, 535**
Schistosoma associated with
Water Infected with, Sterilization of, **351**
*haematobium*
Appendicitis due to, Egypt, **973** *bis*
Complete Cycle of, Experimental Production of, **532**
Intermediate Host found in Nile, 971 *bis*
*japonica*
Appendicitis due to, **972**
Infection with
as Cause of Acute Appendicitis, **536**
Complicated with Amoebic Dysentery, **536**
Serological Studies with, **535** *bis*
*mansoni*
Infection with
in Man and Experimental Animals, Pathology of, **534**
Precipitin Test in, **534, 535**
New Definitive Host for, **533**
Ova of, in Stools: New Detection Method, **971**
of Spleen, and Egyptian Splenomegaly, **971**
Treatment
Antimosan, **532, 533**
Carbon Tetrachloride, **533** *bis*
Emetine, Periodide, 577
Fuadin Injections, Intramuscular, **972**
STRONGYLOIDOSIS, and Idiopathic Hypereosinophilia, **543**
TAPEWORM ANAEMIA
Treatment of, **540** *bis*
TAPEWORM INFESTATION
in Children, Treatment, **975**
Kenya, 407
TRICHINIASIS-TRICHINOSIS
with Diplopia and Polyserositis, **987**
Experimental, Intradermal Reaction in, **551**
Treatment by Carbon Tetrachloride, 970
Helminthic Analysis, Quantitative, **969**
Helminthicides, Rosanilin Derivatives as, **970**
PARASITES, *see also under the various* DISEASES, *supra*
Animal
Found in
Korea, **526**
Street Dust, Johannesburg (1925–1928), **525**
Eggs of, Search for, by Means of Cedar Oil, **528, 969**
Human
Found in
Ankole, Uganda, **525**

**Helminthiasis**—*cont.*
PARASITES—*cont.*
Human—*cont.*
Found in—*cont.*
Belgian Congo: Kasai Mines, **411**
Brazil, **528**
Colombia, Western, 585–6
Madagascar, **526**
Nyasaland, North, **525**
Oceania, 418
Philadelphia, U.S.A., **527**
Turkey, **527**
Yunnan, 416
Unidentified, of the Liver, **529**
Infestation with, Influence of, on Pancreatic Secretion, **528**
Intestinal Worms, Infestation by, in Georgia, **970**
Surgical, **969**
New Helminths from African Animals, **971**
Species, Genera and Families, referred to, by Name
Cestodes, Rare, of Man, **975**
*Cryptocotyle*, Development of, in Final Host, **974**
*Cysticercus fasciolaris*, Effects of, on White Rat, **540**
Diphyllobothrids, *Sparganum* Stage of, Experiments with, **539** *bis*
*Diphyllobothrium latum*
Dog as Reservoir of, **538**
Experimental Studies on, **539**
in Fish and in Man, Northern U.S.A., and Canada, **536, 537** *ter*, **538** *ter*
Infection with, **975**
Cryptic, **538**
*Dipylidium caninum*, Infection in Man, **542** *bis*
*Echinochasmus perfoliatus*, **531**
*Fasciola gigantica*, Infestation by, of Man; Russia, **530**
*hepatica*
Cysts of, Biological Observation of, **529**
Infestation in Man, **529, 530, 971, 973**
Ova of, *Catenaria anguillulae* as Parasite of, **530**
Young Worms, Migration Route of, in Final Host, **529**
*Gongylonema hominis*, Fifth Case of Infestation by, in Man, U.S.A., **557**
Gordiidae, as Parasites of Man, **557**
*Hepaticola*, Infestation by, of Chimpanzee, **556**
*Hymenolepis fraterna*, Development Cycle of, **976**
*microstoma* and *diminuta*, New Intermediate Hosts of, **977** *bis*
Nematodes
*Cyathostoma* genus; Lung Infestation by one of: Manila, **988**
Larvae of, Mature, Capacity of, for Resistance, **981**
*Sparganum mansoni*, What is it? **976**
*Taenia*, Infestation with, with Appendicular Symptoms, **977**

**Helminthiasis**—*cont.*
Tapeworm(s)
Lipoids of, Serological Behaviour of, **541**
Proglottides, Permanent Preparation of, **977**
Transduodenal Expulsion of, **542**
Trematodes from Freshwater Fish, **531**
*Heterophyidae*, Studies on, **974** *bis*
Liver, in Man and Animals, **529, 530, 971, 973**
Ova of, Passage of, by Human Intestinal Tract, **973**
of Snails and Vertebrates; Philippines, **531** *bis*
*Trichostrongylus orientalis*, as Porcupine Parasites, **990**
Titles of Unnoticed Papers, 557–8, 993

**HISTORICAL,** 280–91
Black Death, the, in Danish Folklore and Tradition, **285**
Books on, Reviewed, **345, 427**
Intermittent Fever (Malaria, *q.v.*) in the Rhine Province, and its Disappearance, **280**
Leprosy, Great Epidemic of, in Europe, **286**
Malaria
Forms of, History of, **281**
in Germany, **20**
Mannheim Epidemic, 1761, **281**
in Holland, **282**
in Italy
in relation to the History of Rome, **283** *bis*
Mantua, History of, **282**
Tertiana duplex, 16th Century, 281
Presidential Address, **289**
Rabies in Antiquity, **287**
Relapsing Fever Spirochaete, a Letter on, from Otto Obermeier, the Discoverer of it, **285**
Scabies, History of, Bonomi's Letter to Redi, An Important Document in, **289**
Snake-Bite, Past Methods of Treating, **290**
Syphilis, American Origin of, **284,** 289
Title of Unnoticed Paper, 291
Yellow Fever; Research in, Review of, **289**

**JAPANESE RIVER FEVER,** 52–4
Etiology, **52, 848**
Incidence
Malaya, **848**
Sumatra: Remarkable Case, **53**
Laboratory Infection with, Fatal, **53**
Treatment by Stabilarsan, and Lumbar Puncture, **54**

**Jaundice, Infectious,** *see* **LEPTOSPIROSES**

**Juxta-Articular Nodules,** *see under* **YAWS AND SYPHILIS**

**KALA AZAR** (Leishmaniases, All Forms), 310–30, 740–57
Blood Conditions in
Catalase Diminution, **330**
Serum of
Changes in, **319**
Surface Tension, **318**
CANINE, **755**
Abnormal Parasite Distribution in, **755**
Incidence
Algeria, 755
Central Asia, **323, 324** *bis*
Tashkent, 755 *bis*
Skin Lesions in, **323**
Case of, **743**
Chopra and Gupta Reaction in, Value of, Reservation on, **746**
DERMAL OR CUTANEOUS (Aleppo Button, Biskra Button, Clou de Mila, Delhi Boil, Oriental Sore, Tropical Sore)
Case of Long Incubation, **327**
Facial, **752**
Following Visceral Leishmaniasis, **751**
Giant Cell Reaction in, **328**
Immunity in, **325**
Incidence
Age, 328
Geographical
Algeria, **328,** 748
Brazil, 328
Crete, Multiple, **751**
Egypt: Alexandria, 328
France: Marseilles, 752
Greece: Athens, **327**
French Indo-China
Tonkin, 327
India: Lahore, 752, 753
Turkey: Cesarea, 752
Post-Kala-Azar, Case of, **751**
Prophylaxis: Vaccination, 754
and Visceral: Their Relations, 329
MUCO-CUTANEOUS=ESPUNDIA, **329,** 747, **750**
Incidence
America, Various Parts, **750**
Brazil, **750, 753**
Peru, 329
Spain, **315**
Polypus of, **326**
Psoriasis and, **328**
Transmission by Direct Contagion, **748**
Transmission Problems, **312**
Treatment
Berberine, **752** *ter*, **753**
661 and 471, **327**
Synectol: Cure, **326**
Virus of, Preservation of, in White Mice, **754**
DERMAL LEISHMANOID
Note on, **325**
Diagnosis
Amato's Test in, **746**
Antimony Tests for, Critical Examination of, **318**
Spleen-Puncture Syringe for (*ill.*), **316**
Formol-Gel Reaction in, Causation of, **746**
Hypoproteinaemia in, Significance of, **744**
Incidence
Age, 314, 315

**Kala Azar**—*cont.*
Incidence—*cont.*
Geographical
China, 742 *bis*
Epidemiology of, **741**
Experiments on, **311**
Cirenaica, and other Parts of North Africa, **315**
India
Recent Researches on, **310**
Assam, 751
Lebong, 743
Patna City, Report on Survey, Aug.–Nov. 1923, **313**
Spain, 314, 315 *bis*, **330** *bis*
Cadiz Province, **329**
Tunis, **740**
Indian Commission on (1927–8), Report of, **329**
Leishmania (Herpetomonas) associated with All Forms
Cultivation of, on Solid Media, **747**
in Grey Lizard, Palestine, **757**
*canis*, Survival of, in *Phleb. papatasii*, **755**
*ceramodactyli* n. sp., **756**
*donovani*
and *canis*, Identity of, Question of, **324**
Infection with, of Hamsters, by Oral and Conjunctival Routes, **323**
*tropica*
Animal Experiments with, **753**
Cultures of, Virulence of, for White Mice, **754**
Metabolism of, **329**
Occurrence of, in Wild *Phleb. papatasii*, **749**
Transmission of
Direct from one *Phleb. papatasii* to another, **325**
by *Phleb. sergenti*, 324, **748**
Leishmania Vaccine, Experiments with, **747**
MEDITERRANEAN OR INFANTILE
Incidence
Central Asia
Samarkand, 324
Tashkent, in Connection with the Disease in Dogs, **324**
France, **316**
Paris, in Adult, **330**
Greece; Athens, **316**
Italy, **740**
[Spain], **314, 315**
Methylene Blue, Solution of, Decolourization of, when Left in Contact with Kala Azar Serum, **330**
and Oriental Sore, *q.v.*, *under* DERMAL, *supra*
Transmission Problem, **312**
Phlebotomus Flies in relation to, 310 *et passim.*, 748–9
*argentipes*
Transmission Experiments with, **322**
*papatasii*, Wild, Occurrence in, of *L. tropica*, **749**
*sergenti*
Infection of, with *L. tropica*, **324**
Attempts to Transmit the Organism by, **748**

**Kala Azar**—*cont.*
Protein Graphs in (*ill.*), **319**
with Recovery after Acute Anterior Poliomyelitis, **743**
Research on, Recent, in India, **310**
Rieckenberg Reaction in, **323**
Titles of Unnoticed Papers, 330, 757
Transmission, Experimental of, Feeding Sandflies for, New Technique for, **742**
Transmission Experiments, with *Phlebotomus argentipes*, **322**
Transmission by Sandflies, Slapping as a Factor in, **742**
Treatment
Serological Control of, **744**
Various Methods
Aminostiburea, **321** *bis*
Antimony Compounds, 571
Neo-Stibosan, Intensive, **321**
Stibamine Glucoside (Neostam), **743**
Urea-Stibamine, **743**
*Vitex pedunculata*, Experimental, **322**
Unusual Complication of, **743**
Visceral and Dermal (*q.v.*, *supra*), their Relations, **329**

**LABORATORY REPORTS,** 389–405
Algeria, Pasteur Inst. (1927), 397
Dutch East Indies
Bandoeng: Vaccine Estab. & Pasteur Inst. (1927), 402
Weltevreden: Med. Lab. (1927), 403
Federated Malay States: Inst. for Med. Research (1927), 399
Gold Coast: Med. Research Inst. (1926–7), 389
India, Bombay, Haffkine Inst. (1927), 398
Indo-China, Pasteur Insts. (1925), 401; (1926), 402
Kenya, Col. and Protect., Med. Research Lab. (1927), 395
Léopoldville Lab. (1926), 396
Nigeria: Med. Research Inst. (1927), **390**
Kaduna; African Hosp. Lab. (1927), 391
Lagos: African Hosp. Lab. (1927), 391
Palestine: Lab. Sect., Health Dept. (1927), 403
Roy. Soc. Trop. Med. and Hygiene, Lab. Meeting (1928), 405
South African Inst. for Med. Research (1927), 393
United Fruit Co. Boston
Banes Lab. (1927), 404
Tela Ry. Co. Hosp. (1927), 404
Zanzibar Protect. Biol. Div. (1927), 397

**LEPROSY,** 331–44, 607–25, 1031–44
Alkaline Reserve in, **1043**
Aspects of, **616**
Bacteriology
*Mycobacterium leprae*
as a Cultivable Saprophyte, **343**
Attempted Cultivation of, **616, 620** *ter*
Culture Researches on, **1040** *bis*
Lepra Cell: Origin of, Studied in Cultures, **621**

**Leprosy**—*cont.*
Blood and Blood Conditions in
Blood Groups
Among Estonian Lepers, **1042**
Isoagglutination in, **341**
Blood Serum, Phyto- and Photopharmacological Studies on, **619**
Complement Deviation in, **617**
Complement Fixation in, **341**
and Precipitation Test for Syphilis in Lepers, **1041**
Erythrocytes of, Sedimentation Speed of, **341**
Tests of, **341**
Bone Change in, **1036**
Books on, Reviewed, **347, 425**
Cerebrospinal Fluid in
Value of Examining, **337**
Wassermann Reaction in, **618**
Chaulmoogra Oil, Active Principle of, **1038**
Contagion of, Mode of, **615**
Depigmented Patch in, **1036**
Dermal, Sudoral Function in, **614**
Dermal Leishmaniasis in Combination with, **1036**
Desquamation in, Effect on, of Treatment by Ethyl Esters of Chaulmoogra, **621**
Diagnosis
Bacteriological, **336**
Kahn's Reaction in, **341**
New, Based on the Sero-Electric Reaction, **624**
Problems of, **336**
Rubino's Reaction in, **336**
Early, Obscure Neurological Case Simulating, **1036**
Epidemiology of (*see also* Incidence, *infra*), New Aspects of, **1040**
Eruptions in, Acute, **1035**
Erythema, Nodose, in Leper under Treatment, **340**
Etiology, 615
New Aspects of, **1040**
Possibility of Transference by Insects and Experimental Inoculation of Three Men, **331**
Experimental (Culture and Inoculation), **343**
Experimental in
Monkeys, **342**
White Mice, **342**
Geographical Distribution of (*see also* Incidence, *infra*), **1031**
Haematological and Serological Investigations in, **617**
Human
Identity of, with Rat, **1032**
Transmission of, to White Mouse, **1032**
Immunization Processes, Latent, in, **619**
Incidence
Age: Infant, **615**
Familial, **1035**
Geographical
Argentina, **335, 610, 1034**
Belgian Congo
Kasai Mines, 409, 410, 411
Brazil, **609,** 614
Amazonas, **610**
S. Paulo, How Dealt with, **1035**

**Leprosy**—*cont.*
Incidence—*cont.*
Geographical—*cont.*
British Guiana, **621**
Caspian Area: Spread of, **1034**
Central Asia, **1043**
Bukhara, **609**
Usbekistan, **609**
Ceylon, 613
China, 339, **1033, 1034**
Eradication Problem, **332**
Swatow, **341**
Yunnan, 416
Colombia, **610**
Cyprus, **609,** 1033
Dutch East Indies, 613, 616 *ter.*, 1031
Estonia, **1042**
Europe: Great Epidemic of, **286**
Fiji, 1033, 1035
Formosa, **608**
France: Marseilles, **609**
India, 612, 1033, 1036, 1037 *bis*
Assam, 615
Bengal, 594
Madras, 613
Japan, **610,** 1036
Distribution: Prevention, **1043**
Loyalty Islands, **1033**
Malaya
Kuala Lumpur Leper Asylum: Annual Report, **333**
Manipur State, **1033**
Mexico, **609**
Morocco, **335**
New Caledonia, **334, 611**
Nigeria, Southern, Study of, **334**
Norway: Why Decreasing? **611**
Nyasaland, 1032
Palestine, **609**
Philippine Islands, 331, 1036
Riga, **609**
Siam, **610**
South Africa, with Scalp Lesions, 615
Spain: Guezaua, **1035**
Togoland, British Mandated Territory of, **334**
U.S.A., **607, 608**
Race, 607
International Crusade against, with a View to its Extinction, **1032**
Kahn Reaction in, **341, 1041**
Without Cholesterin, **1041**
Legislation and, **1034**
Lepers in Usbekistan, Care taken of, **1043**
Leprosy, **344, 615**
Leprosy Notes, **331, 613, 1032**
Leprotic Infection of Nasal Mucosa, New Method of Treatment of, **621**
Leprous Erythema Nodosum in, **1035**
Leprous Lesions of the Scalp, **615, 1036** *bis*
Leprous Reaction, the, **1037**
Discussed, **614**
Lipoids of, and Tubercle Bacilli, Comparative Studies on, **1042**
MACULAR in Blacks, **1036**
MACULO-ANAESTHETIC: Treatment by Gold Sodium Thiosulphate, **1038**
Management of, Place in, of Pot. Iod., **1037**
Mixed, Clinical Notes on, 577

**Leprosy**—*cont.*
Nasal Lesions in, Persistence of, **1036**
Nerve Abscess in, **613**
NODULAR
Bacillaemia in, **614**
Treatment with Dmelcos, **624** *bis*
Pathology, 615
in a Peripheral Nerve, **1036**
Pirquet's Reaction in, **616**
and Pregnancy, **336**
Primary Sore of, **613**
Prophylaxis
Campaign against, in India, **612**
Control of, **612**
in Japan, **1043**
in New Caledonia, **334**
RAT
Identity of, with Human, **1032**
Treatment by Alepol, **621**
Rubino's Reaction in, **336, 617**
Sedimentation in, Rubino's Method of, Value of, **617**
Serological Reactions in, **617, 618** *bis*, **1041**
Serology and Immunology, Recent Advances in, **341**
Skin Reactions in, Specific, **616** *bis*
Skin Signs in, **1035, 1036**
Titles of Unnoticed Papers, 344, 625, 1044
Transmission by Insects
Possibility of, 615, **616**
Experiments on, **331**
Transmission of, to Lower Monkeys, **1043**
Treatment, **338**
Brief Instructions for, **339**
History of, **343**
at the Mahaica Leper Hospital, British Guiana, **621**
Sedimentation Test in, Value of, **1039**
Various Methods
Alepol, **621**
B.C.G., **340**
Bacteriotherapy, **339**
Calcium Chloride, Intravenous, **623**
*Calophyllum bigator*, Ethyl Esters of: Results, **1038**
Carbonic Acid Snow and Lopion, **1039**
Chaulmoogra Oil, 1038
Derivatives of, Oral Administration of, **337**
Ethyl Esters of, Effect of, on Desquamation, **621**
Dmelcos Vaccine Effect in Nodular Case, **624** *bis*
Eparseno, **616**
Ephedrine Hydrochloride for Nerve and Joint Pains, **340**
Fibrolysin: Results, **1043**
Gold Sodium Thiosulphate, in Maculo-Anaesthetic Cases, **1038, 1039**
Industrial Therapy, **624**
Malaria Therapy, **623**
Ol. Gynocardium, &c., Four Cures, **622**
Ol. Hydnocarp. and Alepol, **338**
Pot. Iod., **1037**
Intravenous: Results, **622**
Tellurium, **340** *bis*
Various Preparations, **339**
X-Rays, Spinally Applied, **1039**
Urine in, Toxicity of, **1042**

**Leprosy**—*cont.*
VISCERAL, Morphology of, **1043**
Wassermann Reaction in
Non-Specific, Thermal Influence upon, **617**
in Serum and C.S.F., **618**

**LEPTOSPIROSES,** 129–35, 669–73
Leptospirae=Spirochaetae
Cultures of, Pure, Isolation of, by Filtration or Centrifugation, **132**
Species Pathogenic for Mice in Ghent Town Water, **671**
*autumnalis* Types A & B. of Japanese Autumnal Fever, **129**
*icterohaemorrhagiae*
Biology of, in the Tropics, **670**
in Wild Rats; Philippine Islands, **673**
*pseudo-icterohaemorrhagiae*
Agglutinationof (Vincent's Water Strain) in Successive Cultures, **131**
Various
Immunological Relationships of, **130** *bis*
Resistance of, to External Agents, Comparative Study of, **129**
Slime Fever, **672**
in Germany, 1926 and 1927, **132**
Spirochaetoses, Icterogenic, Pathogenicity of, **131, 669** *bis*
Spirochaetosis icterohaemorrhagica=Weil's Disease
of Aquatic Origin, **134,** 135, **671, 672**
Bismuth's Protective Effects against, Mechanism of, 134
Complement Fixation in, **133**
in Guineapigs, Normal and other, Immunization against, **672**
Incidence
Dutch East Indies: Sumatra, **670**
Japan, **133**
Spain: Cadiz, **135**
U.S.S.R.
Moscow, Epidemiological Conditions for, **673**
in a Sailor, **671**
Prophylaxis
Bismuth in, **134**
Japanese, **133**
Recent Publications on, **135**
Surgical Form, **135**
Treatment by, Bismuth, **134, 672**
Title of Unnoticed Paper, 135

**MALARIA,** 1–38, 360–88, 904–51
ACUTE with Cerebral Apoplexy, **13**
and Chronic, Treatment by Quinine Dimethylarsenate, **380**
Anaemia of, and Pernicious Anaemia, Reticulocyte Behaviour in, Difference in, **937**
Anopheles in relation to
Book on, Reviewed, **514**
Habits of, in relation to Control-measures, **919**
Infected, Habits of, **364**

**Malaria**—*cont.*
Anopheles in relation to—*cont.*
Larvae
Density of, table, 361
Found in, Borrow-Pits, Duars Tea Gardens, **909**
Malarious Parasites in, Development of, Effect on, of Heterologous Meals, **948**
in Shantung, Experiments with, **910**
Species Known or Suspect as Carriers
*maculipennis*
Infection of, from a Malaria Patient, Quinine-treated, **28**
*philippinensis*, in Bengal, **8**
*punctipennis*, **31**
*rossi*
Experimental Susceptibility of, for Malarial Infections, **928**
Species Sanitation, Prospects of, in Java, **912**
of Stanleyville, Sporozoic and Zygotic Indexes of, **939**
Transmission by, of the Disease in Ceylon, **947**
Anophelism *sine* Malaria, in the Neighbourhood of Amsterdam, **915**
Aorta in, Dilatation of, **948**
and Appendicitis, **12** *bis*
and Asthma, **12, 370, 927**
AVIAN
Treatment by
Absorption of Chemical Compounds by Erythrocytes and its Significance, in, **24** *bis*
Plasmoquine: Effect on Haemoproteus, **22**
Blood in, *see also* Anaemia, *supra*
Bilirubinaemia in, **26, 927**
Blood Destruction in Chronic Form, **26**
Blood Groups in, Significance of, **10**
Blood Serum in, Lytic Properties of, and its Action on Parasites, **29**
Complement Deviation in, **382**
Gametes in and Oocysts in Mosquito's Stomach, Correlation between, **28**
Leucocyte Formula in, **29**
Leucocytes in Incubation Period of Experimental Malaria, **938**
Leucocytosis in, **936**
Peripheral
and Bone-Marrow, Parasites in, Examination for, **929**
Large Mononuclear Cells in, **936**
Physico-Chemical Changes in, Produced by the Malarial Paroxysm, **381**
Quantity of, in Cases with Enlarged Spleen, Estimation of, **938**
Bone Marrow in, **28**
Books on, Reviewed, **80, 81, 166** *bis*, **427, 428, 956**
Cataract as Caused by, **370**
CHRONIC
Blood Destruction in, **26**
Parasites of, Morphology of, **26, 387**
Treatment, **377**
Coincidence of, and Typhoid Fever (*q.v. under* **ENTERIC FEVERS**), **925**

**Malaria**—*cont.*
Coma of, Sod. Cacodylate for, **919**
Congenital, **11** *quat.*, **387** *bis*
Dakar, 412
Correction, **388**
County Health Depts. for U.S.A. to Handle, **919**
Critical Review of Present Position of, **368**
Dengue in relation to, **842**
Development of, in Native Children, in a Region rendered Progressively Healthy by Anti-malarial Measures, **363**
Diagnosis
Fouchet's Reaction in, **383**
Serological Tests for, **383** *quat.*, **940** *ter*, **941**
Economic Significance of, to a Railway, **368**
Endemic, Measurement of, **362**
Epidemiological Work in, The Splenic Index as a Base of, **10**
Epidemiology (*see also* Incidence, *infra*), **588**
Holland, **916**
Macedonia, **917**
Palestine, **6**
Experimental
Incubation Period of, The Leucocytes in, **938**
Prophylactic Use in, of Quinine and Other Drugs, **923**
Studies in, **944**
Feeding in relation to, **924**
Formol-Gel Reaction in, **942**
Forms of, History of, **281**
Fouchet Reaction in, Abnormal Colour Development in, **383**
Haemorrhagic, **927**
Historical References, **280, 281, 282**
Human, Attempts to Transmit to Animals, **382**
Icterus, Haemolytic, after, **387**
Immunity, Species, **928**
Immunity in, Studies on, **944**
Incidence
Age, **11** *quat.*
Children, African, **11**
Cardiac Disturbances in, and Convulsions in, **926** *bis*
Oedema in, **370**
Class and Occupation
British Troops, East African Campaign, Severe, **367**
Geographical
Algeria, 363 *bis*
Epidemiology of, and Prophylactic Studies, **3**
Prophylaxis of, **946**
Argentina
Tucuman: Relation between Malaria and Rice, **366**
Asia: Georgia, **928**
Recolonization of Malarial Districts in, **365**
Azerbaijan, Republic
Endemicity and Physical Conditions of the Population, **918**
Barbados, **1** *bis*
Brazil, with Lung Changes, **13**

**Malaria**—*cont.*
Incidence—*cont.*
Geographical—*cont.*
British Solomon Islands, Incidence of Different Types, **369**
Bulgaria, **370**
Caribbean Area, Measures of, **2**
Cameroons, French
Endemic Malarial Index in Feb.–Mar., 1928, **947**
Central Asia
Turkestan: Tashkent, 926
Central Kavirondo Reserve, **947**
Ceylon, Transmission of, by Anopheles, **947**
China
Shantung, Experiments with, **910**
Yunnan, 416
Cochin China, **5**
Colombia, Western, 585
Congo, Belgian, 410, 411
Cinchona Cultivation in relation to, **364**
Katanga, 931
Dutch East Indies, **911**
Treatment with Plasmoquine and Quinine, 372, 373 *bis*
Java
East and South Coasts of, **912** *bis*
Nias, Island of, off Sumatra, **912**
East Africa, Severe, in British Troops in the Campaign in, **367**
Federated Malay States
Kuala Lumpur, 931
Research Division, Report of (1927), **360**
France
Marseilles, contracted in Syria or Morocco, 934
Meuse Department, **12**
French Guinea
Upper Volta, in
Europeans, **2**
Natives, **2**
Germany
Mannheim Epidemic of 1761, **281**
Rhine Provinces, Disappearance of, **280**
Greece: Athens, 926
Haiti, **922**
Holland, **916**
Distribution of, **915** *bis*
History of, **282**
Report on, of Malaria Commission for 1927 and 1928, **915**
India, 13 *bis*
Andaman Islands, **5**
Assam, in Tea Gardens, **4**
Bengal
Calcutta, Environs of, Malaria Survey of, Report, **3**
Duars Tea Gardens, **909**
Krishnagar, **8**
Malda District, Site of Gour; Flood and Flush Systems, **5**
Sonapur Field Observatory, Report of, 1921–25, **3**
Wassermann Test in, 593
Birnagar, Problem of Control, **909**
Bombay, **4**

**Malaria**—*cont.*
Incidence—*cont.*
Geographical—*cont.*
India—*cont.*
Coorg, **362**
Madras
Surveys of Mopad and other Areas, **909**
Italy
Bologna District, 372
Ferrara Province, **913**
Maccarese, **914**
Mantua, History of, **282**
Rome, with Asthma, in Child, **12**
Factor in Roman History, **283**
Jamaica, 13
Kenya, 407, 925
Lumbwa Reserve Epidemic, 1928, Report on, **394**
Teita District, **907**
Uasin Gishu and Trans Nzoia, **907**
Macedonia, **917**
Madagascar, **949**
Diego Suarez Province: Spleen Indexes, **387**
Malaya, 374
Morocco
in a Red Cross Hospital, **370**
Rif Region, **3**
Palestine, **6, 947**
Malaria Survey and Research Unit in, Reports of (1927), **365** *bis*
Hedera, 922
Hule, **364, 382**
Philippine Islands,
Transmission in, **367**
Olangapo; in Civilians, **361**
Porto Rico
Coincident with Typhoid Fever, **925**
Incidence and Problems of, **1** *bis*
Report of Bureau of Malaria Control (1926–7), **366**
Portuguese West Africa
Loanda, Malarial Index of, **363**
Rabaul, 583–4
Rhodesia, Southern, Endemic and Epidemic, **908**
Roumania: Immunity in, 945
Singapore: H.M. Naval Base, **6**
Spain
Aragon, Estanca de Alcañiz, **917**
Viscaya; Indigenous, **7**
Trans-Jordan: Malaria Survey, **6**
Trinidad, Pitch Lake Region, **8**
Uganda, 948
U.S.A.
Southern States, 919
South-western States, Irrigated Regions of, **921**
Tennessee, Flooding in, **921**
U.S.S.R., Lower Volga Region, Book on, Reviewed, **81**
Voronezh (1924–28), **918**
Venezuela, **921**
Studies in, **366**
Race, **11,** 382, 927
Bedouins, **922**
Untreated Indigenous Population, **364**
Incubation Period in, **9**

**Malaria**—*cont.*
as an Independent Etiological Factor in Portal Cirrhosis, **368**
Inoculation Malaria
Anti-syphilitic
Spleen Rupture after, and During, Treatment, **386, 946**
Additions from, to Knowledge, **30**
Biological Studies in, **385**
Blood in, of G.P.I. Patients, **386**
Blood Groups in
and Course of, **386**
Influence of, on Incubation, **384**
Significance of, **10, 943**
Clinical Notes on, **385**
Experience with, **385**
Incubation of, Climate and Blood Groups as Influencing, **384**
Intraspinal Injection of, **381**
and Natural, Initial Fever and Incubation in, **30**
Natural Transmission of, **945**
Parasites in, **30**
Life Cycle of, Clinical Significance of, **386**
Pathogeny of, **385**
Technique of, **946**
Treatment by, Centres for Inauguration of, Suggested, **947**
Kidney Lesions due to, **926**
Lung Changes in, **13**
Malaria Commission of the Health Organization of the League of Nations, Report of the Work of, **904**
Malaria Survey, a, How to do, **360**
Malaria Surveys
in Haiti, **922**
of Mopad and other Areas, Madras (1926–7), **909**
Malaria-Mosquito Survey of the Duars Tea Gardens, 909
Malaria Therapy, **30**
Malarial Attack, Not due to Haemoclastic Shock, **31**
Mosquitoes associated with, *see* Anopheles, *supra*
New Serological Reaction in, **942**
Parasites (Plasmodia)
Biology of in Infection with Virulent Blood, **928** *bis*
of Chronic Malaria, Morphology of, **26, 387**
in Compatible and Incompatible Serums, Research on, **943**
Cultivation of, **936**
Aerobically, Simplified Technique for, **383**
Crescents in, Evolution and Degeneration of, **935**
Merozoites seen in, Fate of, **935**
Effects on, of Quinine, **23**
Gamete-Formation in Cultures, **381**
Gametocytes of, Viability of, Effect on, of Small Doses of Plasmoquine Measured by Mosquito, Infection Experiments, **939**
Human, Inoculation of, into Animals, **29**
in Inoculation Malaria, *see under that head, supra*

**Malaria**—*cont.*
Parasites (Plasmodia)—*cont.*
in Peripheral Blood and Bone-Marrow in, Examination for, **929**
Unusual in Character, **382**
*falciparum*, Relation of, to the Human Red Blood Cell, as Determined by Sections, **381**
*tenue*, Review of Literature and Case Report, **8**
*vivax* and *falciparum*, Factors of Geographical Distribution of, **9**
in Madagascan Natives, Action on, of Quinine Stovarsol, **949**
Perniciousness in, Book on, Reviewed, **80**
Pigment, Malarial, Origin of, **29**
Practical Review of, **924**
Precipitin Tests in, 27, **27, 942**
in Pregnancy and Labour, **924**
Pregnancy and, and Quinine in the Tropics, **37**
Problems of Treatment of, Guide for Government Officials, etc., in, **32**
Prophylaxis
Antimalaria and Anti-Anopheles Measures, 924
in Algeria, **946**
Anopheles Species Sanitation (Proposed), Java, **912**
Australian, **37**
Bureau of Control, Report of, 1926–7, **27**
Control Problem, at Birnagar, **909**
Guide on, for Officials, etc., **32**
Italy, **913, 914** *bis*
Campaign against, 1926–7, Reports on, **34** *ter*
Larvicidal Fish in Shantung, **910**
Larvicides
Paris Green, **34, 583**
Stoxal, **583**
Malayan : Report, 1927, **36**
at Mombasa, **35**
Mosquito Control
Practical Measures of, **33**
in U.S.A. and Canada, **37**
Mosquito Proofing of Barracks ; Further Results, **581**
in Palestine, **947**
at Panama, **36**
Paris Green, New Way of Distributing, **920**
Tests of, for Required Minimum Efficiency, **920**
Plasmoquine in, **933**
in Bedouins, **922**
among Russian Peat-bog Workers, **36**
at Quantico, Va., **35**
Quinine and other Drugs in Experimental Cases, **923**
Results at Barnaul from 1921 to 1927, **369**
Sanitation, Antimalarial, Principles of, **363**
Screening, **921**
Studies on, in Leflore County, Miss., **33**
Shading for Anopheline Areas, **369**
Twenty-five Years of Control, **31**

**Malaria**—*cont.*
QUARTAN
Incidence: British Solomon Islands, **369**
Parasite Picture and Leucocyte Formula of, during Quinine Treatment, **924**
Treatment by Plasmoquine, **931**
Quinine, *see also under* Prophylaxis, *and* Treatment
Influence of, on Urobilinogen Output, **384**
and other Drugs, Prophylactic Use of, in Experimental Cases, **923**
Quinine Salts, Solubility of, Effect on, of Alkalis, **929**
Quinine Stovarsol, Action of, on *P. vivax* in Madagascan Natives, **949**
Reactivating Function of, **31**
Reduction of, after an Antimalarial Campaign, Estimation of, **369**
Relapse, 6 Years after Leaving Country of Infection, **948**
Report of Bureau of Malaria Control, 1926–7, **27**
Reticulo-Endothelial System in, **936**
Rice Cultivation in relation to, **366**
Sidelights on, **10**
Simulating Confusional Insanity, **13**
Soil Content, Nitrogenous and Total Organic, in relation to, **368**
Species Immunity in, **928**
Spleen in, Alterations of, **926**
Spleen-rate and Parasite-rate as Measures of Malaria; Caribbean Area, **2**
Spleen Rupture in: Splenectomy: Recovery, **13**
Traumatic, **948**
Splenic Index as Base of Epidemiological Work in, **10**
Studies in, **922**
with Special Reference to Treatment, **929**
in Venezuela, **366**
Study of, New and Old Problem of, **9**
SUBTERTIAN, Aestivo-Autumnal, Malignant Tertian, or Pernicious
Carriers of, Treatment of, by Plasmoquine Compound, **931**
Clinical Features, **13, 14**
Cure, Natural and Therapeutic, **25**
Incidence
China: Shantung, **13**
Dutch East Indies, 927
Interesting Case of, **13**
in Parturient Woman, **387**
Pathology, **9**
Prophylaxis, **9**
Treatment, **9**
Quinine, Intravenous, **22**
Symposia on: U.S.A., **7, 919**
TERTIAN, BENIGN
Accidental, **927**
Chronic
Relapse Rate in, Effects on, of Diet Rich in Sugar, **10**
Treatment by
Quinine-Stovarsol, Oral, **379** *ter*
Quinine-Troposan, **380**
Development in, of Sporulating Forms, Double Infection, and Parthenogenesis, **25** *bis*

**Malaria**—*cont.*
TERTIAN, BENIGN—*cont.*
Gametocytes of, and Early Appearance of, in Cases Transmitted by *A. punctipennis*, **31**
in India: Kasauli, 929
Infection of, Resistance to, **945**
Inhibiting Effects on, of Fresh Infection with Syphilis, **946**
Tertiana duplex, 16th Century, 281
Treatment
Cinchona Alkaloids, **929**
Sulfarsenol, **935**
Titles of Unnoticed Papers, 37–8, 387–8, 949–51
Transmission, **367**
Treatment
Studies in, **929**
Various Methods
Aseplene, **380**
Autohaemotherapy, **949**
Cinchona, Total Alkaloids, **23**
Dimeplasmin, **931** *bis*
Dimethylarsenate of Quinine, **380**
Euquinine, Drugs of the Type of, Relative Therapeutic Efficacy of, **378**
Plasmoquine, **532, 930, 931, 932, 933** *bis*, **934** *bis*
Clinical Results, **16**
Detection of, in Urine, **375**
for Carriers, **931**
in the Field, **374**
Observations on, **934** *bis*
and Plasmoquine Compound, **14, 15** *bis*, **16** *sept*, 18 *bis*, 19 *quat.* **20** *ter*, 21 *quat.* **22, 370, 371** *bis*, **372** *ter*, **373** *ter*, **374** *bis*, **375** *bis*, **376** *bis*, **377** *ter*
Secondary Effects, **376** *bis*
Toxic Effects, 14, **16, 377**
Unsuited to Subtertian Cases, **919**
Quinine, **919**
Compared with Plasmoquine, **19**
Early, **23**
Effects of, on Parasites, **23**
Excretion of, in Human Mother's Milk, **24**
Influence of, on Urobilinogen Output, **384**
Intramuscular, **22**
Intravenous, Limits to Use of, **378**
with Plasmoquine, **16** *bis*, **373**
Stock Solutions, **23**
Quinine dimethylarsenate, in Acute and Chronic Forms of the Disease, **380**
Quinine-Stovarsol, Oral, **379** *ter*
Quinine Troposan, **380**
Quinetum, Cinchonine, and Quinidine, **930**
Quinetum, with Plasmoquine, **374**
Smalarina: Useless, **25** *bis*
Sod. Cacodylate in Coma, &c., **919**
Stovarsol, **24**
Stovarsol Quinine, **934**
Tropical Asthma as Malarial, **370**
Urinary Creatin in, **948**
Urobilinogen Output in, Influence on, of Quinine, **384**

**MEDICAL ZOOLOGY** (*see also under Diseases concerned*), 227–258, 259-279, 519–524, 770–781, 785–824
Abridgements, Bare titles, not separately indexed, 258, 824
Acari, Zoological Studies on, **268**
*Aëdes desbansi*, Biology of, 814
*aegypti*, in Adriatic and S. Italy, **254, 255**
in Dutch East Indies, **254**
in Brazzaville, Breeding Places, **813**
Larval Stage, **243**
in India, in reference to Dengue and Yellow Fever, **254**
*notoscriptus*, Breeding Places, Queensland, **809**
*Alysia manducator*, Parasite of Muscoid Flies, **816**
Amoebae, of Cockroach, Cultivation, **231**
Human, Morphology, **797**
Intestinal, Differentiation, **230**
with four-Nucleated Cysts, **230**
*Ancistrodon blomhofii*, Toxicology of Venom, **777**
Anopheles, of Ceram Island, **814**
of Ceylon, 241
Dutch India, Precipitin Test for Blood, **251**
in Formosa, Distribution and Habits, **249**
Indian, Classification of, **250**
Baroda Camp, **811**
of Hazaribagh, **809**
of Kenya, and Malaria, **248**
of North America, Bionomics of, **810**
*Anopheles*
*bachmanni*, **254**
*fuliginosus*, *pallidus* and *philippinensis*, Larvae of, **815**
*gambiae*, in Kenya, 248
*jeyporiensis*, Larvae of, **253**
*longirostris*, n. sp., **253**
*maculipennis*, Larva and its Ecology, **243, 251**
Larval Development, **252, 815**
var. *atroparvus*, **815**
of Myzorhynchus Group, Dutch East Indies, **253**
Nyssorhynchus Group, Variability, **252**
*pseudopunctipennis*, Blood Feeding Habits, **253**
Range of Flight, **815**
*rossii*, Receptivity to Malarial Infection, **812**
*superpictus*, Biology of, in Serbia, **252**
Anopheles, Biology, Variation of Water Reaction, **812**
of Electrical Conductivity of, **814**
of Oxygen, **814**
Hosts for Parasites of Bird Malaria, **253**
Larvae, and Classification of Indian Mosquitoes, **250**
Contractile Thoracic Appendages, **250**
Nutrition of, **815**
Preservation and Mounting of, **806**
Migration and Dispersion of, **811**
Modification by Environment, **250**
Race Question, **250, 814**
*see also* Mosquitoes
Antivenin Institute of America, Bulletin, **268, 270, 779**
Development of, United Fruit Co., **271**

**Medical Zoology**—*cont.*
Antivenomous Sera, Study of, **780**
Arachnoidism, Case of, **821**
Balantidiasis, in Man, **240** *bis*
*Balantidium coli* in Man and Pig, **239**
Barracuda, Dangerous to Man, **791**
Bartonella Anaemia of White Rats, **802**
*canis*, Infection of Dog by, **803**
*muris-ratti* and Infectious Anaemia of Rats (Critical Review), **519-524**
Bee Sting, Anaphylaxis from, **277**
Beetle, Coprid, Infestation of Human Gut, **265**
Paederus, as Cause of Dermatitis, **821**
Toxic Properties of, **264, 265**
*Blastocystis hominis*, Cases of Enteritis, **803**
*Bodo urinarius*, Parasitic in Urine, **800**
*Calliphora erythrocephala*, Larval Stage, **243**
Carmine Stains for Intestinal Protozoa, **230**
Caterpillar, Hairy, Venom of, **277**
Charophyta, Larvicidal Properties Alleged, **246**
*Chilomastix aulastomi*, Culture of, **798**
*mesnili*, in Gall Bladder, **228**
*Chrysomyia macellaria*, causing Myiasis, **257**
Ciguatera, Cases of, **791**
Coccidia, Specificity of, **238**
Coccidiosis in S. Paulo, **238**
Coprological Statistics, Venezuela, **227**
Crithidia, in Phlebotomus, **799**
Ctenopsyllus, Application of Name, **820**
*Culex fatigans*, Breeding Habits in Queensland, **808**
*hortensis*, Mycosis of Larva, **813**
*pipiens*, Autogenous Cycle of Winter Generations, **808**
Effect of Active Constituents on Skin, **247**
of Argentina, **248**
Culicidae of Belgian Congo, **813**
*Denisonia superba*, Venom of, **774**
*Diemictylus viridescens* in Mosquito Control, **813**
*Dientamoeba fragilis*, in Argentine, **232**
Diptera, Larval, Influence of Starvation on Ovarian Tubules, **243**
Endolimax, in Stools of Case of Enteritis, **795**
*Entamoeba coli*, Haematophagous, **232**
*dispar* Infection, **230**
*histolytica*, Infectivity of Starch-fed Strain, **796**
Life History in Cultures, **795**
Entomologist: Medical, Report for Ceylon, **241**
Entomology
of Antimalarial Measures, Belgian Congo, **249**
of Canton Delta, **242**
of Italian Colonies, **244** *bis*
Fish as Hosts of Helminths, **791**
Flagellates, in Blood of Malarial Case, **800**
in Human Lung, **237**
Fleas on Rodents and Insectivores in Nigeria, **264**
Rat, and Plague in Shanghai, **264**
in Semarang, **820**
Survey in San Juan, Porto Rico, **264**
of Southern Ukraine, **820**
Flies, Bloodsucking, Forcible Feeding of, **818**

* The MEDICAL ZOOLOGY Section of this Index has been prepared at the Bureau, and a different system from that adopted for the other Sections has been followed.

**Medical Zoology**—*cont.*
*Giardia intestinalis*, **235, 795**
*Glossina morsitans*, in Nuba Mountains, Sudan, **819**
Smoke Control, **819**
Southern Rhodesia, Distribution, **260**
Species, Probable Limits, Zululand (*map*), **261**
*see also* Tsetse
Harvest Bug, Present Knowledge of, **267**
Histoplasmosis, Pathology of, **804**
Review of Disease, **803**
House-Flies, Studies in Breeding, **257**
*Hydrus platurus*, Venom of, **781**
Hymenoptera, Stings from **820**
Ichthyology, Medical, **791**
Insect(s) Bites, Antistreptococcal Serum for, **244**
Sensitization to, **804**
Important in Public Health, Catalogue, **241**
Preservation of Haemophilic Viruses, **242**
Insect-borne Diseases in Montana, **244**
*Isospora belli*, Infection in Man, **238, 800**
*hominis* Infection, **800**
Ixodidae of Italian Colonies in Africa, **266**
Laboratory Studies, Fernando Po, **785**
Peking Union Medical College, **786**
*Lachesis gramineus*, Effects of Venom, **777**
*Lamblia intestinalis*, see Giardia
*Latrodectes mactans*, Venom of, **278, 780**
*Leptomonas ctenocephali*, Culture of, **236**
Limnaeidae, Resistance to Desiccation, **790**
*Loxosceles rufescens*, Bite by, **821**
*Lucilia serricata*, Coupling and Oviposit, **816**
Effect of Food on Sex and Size, **243**
Hibernation of, **817**
Malaria, Avian, Anopheline as Host, **253**
Asexual Cycle, Induced Variations, **800, 802**
Infection and Immunity in, **802**
Relapse in, **801**
Studies, Cytological, of, **239, 802**
*Megalopyge urens*, Venom of Larva, **277**
Millipedes, Effect of Bites, **277**
Mites in Human Faeces, **268**
Larval Trombidiid, attacking Man, **267**
Pathological Significance, **822**
Scrub-Itch, of North Queensland, **822**
Zoological Studies, **268**
Mosquitoes of Argentina, **248**
of Belgian Congo, **813**
of Cochin China and South Annam, **248, 805**
of Italian Colonies, **248**
of Shanghai, **810**
of South Africa, **805, 809**
of Tashkent, Hibernation, **813**
Mosquito(es), Breeding Places
Composition of Water, **246, 806**
Crab Burrows as, **807**
Control, General or Special ? **807**
Spotted Newt as Agent, **813**
Ecology, Physical Factors in, **245, 246**
Flies and Rats ; Measures against, **813**
Larvae, Abnormal, **247**
Insect and other Enemies, **246**
Method of Mounting, **805**
Predacious, Delayed Metamorphosis, Possible Application, **808**
Larvicidal Power of Fluorescent Substances, **247**
Notes on, **812**

**Medical Zoology**—*cont.*
Mosquito(es), Breeding Places—*cont.*
Salivary Glands, Virulence of, **247**
Shade as Measure against, **245**
in Relation to Disease, **244**
Yeasts in Oesophageal Diverticula, **247**
Zoophilous and Androphilous Competition, **807**
*Musca domestica*, Studies in Breeding, **257**
Muscoid Maggots, Hymenopterous Parasites of, **876**
Myiasis, Dermal, by Chrysomyia, **818**
by *Phormia regina*, **818**
by Wahlfahrtia, **257**
Nasal, by Chrysomyia, **257**
Palpebral, **818**
*Notechis scutatus*
Bite, Case of, **776**
Venom of, **774**
Organism, Doubtful, in Human Blood, **241**
Ornithodorus, Breeding Management of, **822**
in Earths of Porcupines and Foxes, **823**
New, of Algerian Sahara, **266**
*papillipes*, Effect of Bite, **823**
Studies in Central Asia, **266**
*venezuelensis*, Description of, **266**
*Paederus fuscipes*, Toxic Properties on Skin, **264, 821**
on Rabbit's Eye, **265**
Parasites, in Foochow Area, China, **786**
Intestinal, in North China, **786**
in Philadelphia, **787**
in Jews in Argentine, **788**
Metazoan, from Zululand, **786**
and Problems of Distribution, **785**
simulating Appendicitis, **789**
in Venezuela, **788**
Parasitic Infestation and Diarrhoea in Porto Rico, **787**
Parasitology, Lab. Peking, Report of, **786**
in Tropics, Importance of, **789**
Phlebotomus, Asiatic Species, Synonymy, **255, 815**
*barraudi*, n. sp., **815**
of Bokhara, **257**
*chinensis*, in Syria, **256**
*clydei*, n. sp., India, **256**
Comparative Anatomy of, **257**
*dubosquii*, ? A Valid Species, **256**
Identification, Improved Methods, **255**
*minutus*, Crithidia occurring in, **799**
*papatasii*, Obligatory Hibernation, **256**
Systematic Characters, Variability, **816**
*Phytomonas davidi*, in Euphorbia, Trinidad, **237**
*Plasmodium cathemerium*, Variations in Cycle, **802**
of Cercopithecus, West Africa, **238**
*kochi*, Male and Female Gametocytes, **239**
*Porocephalus armillatus*, **268**
Biology, and Histology of Lesions, **823**
Infection, Case of, **268**
Species in Native of South Africa, **824**
Protozoa, Blood, Immunity in Infections by, **794**
Intestinal, of Armenians, **227**
of Bile Passages, **228**
Detection and Differentiation of, **230**
of Domestic Pig, **230**
Flagellated, Conditions of Presence, **232**

**Medical Zoology**—*cont.*
Protozoa, Blood—*cont.*
Intestinal—*cont.*
of Monkeys, **229**
Relation to Certain Diseases, **794**
of Sweden and Finland, **227**
of Venezuela, **227**
Method for Fixing and Preserving, **789**
of Monkeys and Man, **229, 795**
of Mouth, **793**
Toxins and Venoms, Effect of, **793**
Protozoal Community, Reaction to Organic Acids, **794**
Infestations, Activation by Desplenation, **795**
Treatment, Present Day, **228**
by Stovarsol, **792**
Protozoiasis, **228**
*Prowazekia asiatica*, Human Infection, **237**
*Pseudechis guttatus*, Venom of, **774**
Scorpion Venom, Poisoning by, **276** *ter*
treated by Tutocain, **823**
*Sergentella hominis* (near) in Bloody Urine, **240**
Snake(s), Australia, Dentition and Biting Mechanism, **770**
from Santa Marta, Colombia, **271**
Venoms, of Colombia, **272**
Bite, in Australia, **770**
Symposium on, **773**
in Central America, **271**
Rattlesnake, **273** *bis*
Tiger Snake, **776**
Treatment, **273, 780**
by Ligature, **775**
Subcutaneous-Intravenous Index, **775**
by Massage, **274**
by Serum Therapy, 274, 778
*see also* Antivenin Institute, *and* Antivenomous Sera
Snake Venom,
Adder, Immunity to, of Slow-worm, **275**
Cobra, Neutralizing Power of Soap, **274**
Adsorption by Charcoal, **275**
Copperhead, Australia, **774**
of Formosa, **778**
Javanese Green Snake, **777**
Mamushi, **777**
of North America, **779**
Sea Snake, **781**
of Siam, Experiments on, **275**
Viper, and Antivenoms, **778, 779**
and Experimental Rabies, **780**
Yields in Australia, **773, 774**
Snake, *see also* Antivenin
Sphyroena, Poisoning by, **791**
Spider Bite, Case of, **821**
Venom of, **278**
Stegomyia, *see* Aedes
Stone Fish, Venom of, **278, 279**
*Synanceja horrida*, Venom of, **278, 279**
Termites, Transfaunation of, **790**
Tetrodon Toxin, Influence of Drugs on, **792**
*Theobaldia incidens*, Effect of Food on Sex and Size, **243**
Thysanura and Parasitism, **821**
Tick Paralysis of Italian Colonies, **265, 266**
Titles of Unnoticed Papers, 258, 824

**Medical Zoology**—*cont.*
Tricercomonas of Pig, **798**
Trichomonas, Cultivation of, **232**
in Infant, **235**
in Kitten, **235**
in Rats on Different Diets, **234**
Specificity, Mechanism of, **797**
in Tissue Cultures, **235**
*vaginalis* Infection, **797**
Tritrichomonas, Human, in Cultures of Bacteria, **233**
Separation from Bacteria, **233**
*fecalis*, Cultivation in Tap Water, **233**
Suitability of Foods for, **234**
*Trombicula hirsti*, as Carrier of Tropical Typhus, **822**
*Trypanosoma lewisi*, in Guineapig, **799**
Trypanosomes, Nucleus Division, **799**
Trypanosomidae, Notes on 3 Species, **236**
Tsetse, Areas, Reclamation, Efficacy of Arsenic, **262**
Belt in Western Uganda, **819**
Control Scheme, Nyasaland, **260**
Digestion, Processes of, **263**
Investigation, Nigeria, **259**
in Nuba Mountains, Sudan, **819**
in Southern Rhodesia, **260**
Viper, Action of U.V. Rays on Serum of, **274**
*see* Snakes
Venom of *Notechis scutatus*, **774**
Organs producing, **790**
*see* Snakes
*Xenopsylla astia* in Semarang, **820**

**MISCELLANEOUS,** 406–24, 559–609, 1045–8
Abortion in relation to Depopulation in Oceania, 416, 419
Abscess, Pulmonary, Non-Amoebic, Treatment by Emetine, **595**
*Aëdes taeniorhynchus*, Abatement of, at Chacachacare, **581**
Anaemia, *see under* Blood, *infra*
Asthma, Bronchial, in Yunnan, 416
Bacteriological References
Agglutination, Non-Specific, by Lactic Acid, **574**
Bacillus *Proteus* sp., Case of Human Infection by, **597**
Bacteria
Asporogenous, Sealed Cultures of, Duration of Life of, **576**
*Heliconema vincenti*
Cultivation of, **574**
Morphology and Biology of, **575**
*Pasteurella*, Urethritis due to a, **578**
Staining Methods, **560** *bis*, **562, 563** *ter.*, **564**
Vibrios, of Volga Water, **579**
Bilivaccines, The Question of, **576**
Blood and Blood Conditions
Anaemia of Pregnant Women; Bengal, 594
Blood Cells, Adsorption by, of Quinine, **569**
Blood Changes in Favismus, **594**
Blood Conditions, D.E.I., 413
Blood Counts, Accuracy in, **561**
Counting Chamber, Peroxidase Method for, **561**

**Miscellaneous**—*cont.*
Blood—*cont.*
Blood Counts—*cont.*
Differential, Accuracy Obtained by Various Methods, **561**
Eosinophile, Two Methods, **562**
Techniques for, **561** *ter*, **562**
Blood Films
Making and Fixation of (*fig.*), **559** *bis*
Staining of, Temperature and, **560**
Blood Parasites, Staining of, Rapid, 560
Blood Stains
Rapid Giemsa, **562**
Romanowsky, New Method with, **563**
Vital and Non-Vital Method, Combined, for Staining Corpuscles, **564**
Blood Transfusion : Calcutta, 594
Eosinophilia, Clinical Significance of, on a General Medical Service, **592**
of Goanese, Haemolytic Power of, **562**
Haemolytic Reaction, the, in Enteric and Undulant Fevers, **596**
"Burning" Feet in Malaya and Burma, **423, 424**
Calculus, Vesical, in Yunnan, 416
Cardiac Diseases, Valvular, in African Natives ; Kenya, **593**
$CO_2$-Tension in Alveolar Air, Daily Fluctuations in, in Europe and in the Tropics, **578** *bis*
Clinical Notes, **577**
Countries and Places referred to, Diseases, etc., Prevalent in
Africa, *see also* Parts, *under Names*
Rural, Natives of, Medical and Sanitary Services for, Provision of, **406**
Australia
Commonwealth of
Australian Inst. of Trop. Med., Townsville, **585**
Department of Health, Division of Tropical Hygiene, **583**
Tropical, Queensland
White Field Workers in, **414**
Belgian Congo
Kasai Region
Diseases Prevalent, 409, 410, 411
Kasai Mining Companies, Medical Service of, **409, 410**
China : Yunnan
Kiulungkiang, Diseases Prevalent, **416**
Colombia, Western, Tropical Medicine in, **385**
Dutch East Indies
Blood Conditions, 413
Diseases Prevalent, 413, 589
Queen Wilhelmina Jubilee Inst. : Report (1927), **415**
Toxic Plants in, **565**
Bandoeng
Queen Wilhelmina Hospital for Eye Patients, **415**
Egypt
Lymphadenoma in, with Relapsing Pyrexia, **597**
Splenomegaly in, **591**
Splenectomy for, **590**

**Miscellaneous**—*cont.*
Countries, &c.—*cont.*
Egypt—*cont.*
Alexandria ; Urethritis, Pasteurella-caused, 578
Erithraea, Latah in, Focus of, **581**
Espiritu Santo Island, New Hebrides
Depopulation in, and its Causes, **416**
French Cameroons, N'Sou, Native Poison in, Composition of, **591**
Germany
Hamburg, Inst. at, of Ship and Tropical Maladies, **414**
India
Ether as Anaesthetic of Choice in, **580**
Plants of Medicinal Value in, 566 *bis*
Wassermann Test in, **593**
Kenya
Diseases Prevalent, 406, 407
Rural Areas ; Med. and San. Provisions for Natives, **406**
Valvular Heart Disease in African Natives, **593**
Malaya
"Burning Feet" in Estate Medical Practice, **423, 424**
New Caledonia
Depopulation of, Causes of, Study of, **419**
Pacific, South, Medical Conditions in, **417**
Panama, Canal Zone and City
Diphtheria Control in, **591**
Port-of-Spain
Colonial Hospital, Surgical Work at, Review of, **588**
Porto Rico
Infant Morbidity and Mortality in, **586**
University of ; School of Trop. Med. of, Review of Research (1926–8), **586**
Portuguese Africa, Medical Services in, **408**
Senegal
Dakar
Child Welfare Agencies at, **411**
Diseases Prevalent, 411, 412
Inst. of Social Hygiene at, **412**
Trinidad
Rural Sanitation and Hygiene in, **581**
Tonkin, Typhus Fever in, **589**
Tropics
Tropical Field, The ; its Possibilities for Women, **583**
Tropical Neurasthenia, **413**
Tropical Pathology, Significance of, for Study of Cosmopolitan Diseases, **413**
Tropical Pyomyositis, **589**
Uganda
Diseases Prevalent, 412–13
Maternity Training School, the Lady Coryndon, Namirembe, Report (1927), **412**
U.S.S.R., Vibrios of Volga Water, **579**
Venezuela
Report of Venezuela Gulf Oil Company's Hospital (1927), **588**
Diseases Imported by Europeans into Pacific Islands, 416, 418

**Miscellaneous**—*cont.*
Elephantiasis, Surgical Treatment of, **599**
Ether by Open Method as Anaesthetic of Choice in India, **580**
Favismus, Blood-Changes in, **594**
Goitre in Yunnan, 416
Hyaline Bodies, Intra-cellular, in Some Tropical Affections, **573**
Infectious Diseases, The Reticulo-Endothelial System and, **420** *ter*, **421, 422** *bis*, **423**
Influenza in Espiritu Santo, 416
Jaundice, After Intravenous Treatments, 599
Latah, Focus of: Erithraea, **581**
Lathyrism, Studies on, **596**
Latrine, Practical Bored Hole: Construction details (*ill.*), **1045**
Lymphadenoma, with Relapsing Pyrexia, **597**
Manioc, Fresh, Fatal Asphyxia from, **573**
Measles
Incidence
Espiritu Santo, 416
Yunnan, 416
Medical Services in
Belgian Congo; Kasai Mining Companies, **409, 410**
Portuguese Africa, **408**
Kenya, **406**
Melioidosis, Disease Common to Man and Animals, **599**
Microscopy, *see also* Blood Films, Blood Parasites, *and* Stains
Liquid Paraffin Replacing Oil in, **564**
Microscopical Equipment for Use in the Tropics, **575**
Mumps in Espiritu Santo, 416
Neurasthenia, Tropical, Indications of, 413
Oil Drums, New Use for, **383** (*figs.*), **584**
Pharmacology
Aconite, Indian Varieties of; Chemical Composition: Biologfcal Assay, **566**
Antimony Compounds,
Action of, on the Adrenals, **571**
Organic, Antimony in, Estimation of, **569**
Pentavalent, in Tropical Medicine, **569**
Carbon Tetrachloride, Toxic Symptoms after Administration of, **572**
Cinchona Alkaloids, Action of, on
Free-living Organisms, **568**
Uterus, **567**
Cinchonidine and Cinchonine, Action of, on the Heart, **424**
Drugs, Adsorbed, Intestinal Therapeusis by, **571**
Emetine, Action of on
Gastro-Intestinal Tract, **564**
Rabbit's Heart, **565**
Ephedrine from Indian Varieties of *Ephedra*, **566**
Glutathione, Rôle of, in Production of Trypanatoxyl and Bismoxyl, **579**
Mercurochrome 220 Soluble, **567**

**Miscellaneous**—*cont.*
Pharmacology—*cont.*
Quinine
Adsorption of, by Blood Cells, **569**
Quinine Intolerance, **569**
Stovarsol Poisoning, Case of (*chart*), **572**
Tryponarsyl Poisoning, Case of, **573**
Urea-Stibamine; Preparation: Composition, **569**
Yatren 105, in Treatment of Intestinal Diseases in the Tropics, **577**
Plants, Toxic, Dutch East Indies, **565**
Poisons
Arrow Poison from Colombia, Acting on Heart, **598**
Books on, Reviewed, **168, 348**
N'sou, Native Poison, Cameroons, Composition of, **591**
Pyomyositis, Tropical, **589**
Rheumatism, Acute, in Yunnan, 416
Skin Colour, **576**
Spleen, the, Functions of, **414**
Enlarged, Spontaneous Rupture of, **599**
Tetanus in Children
Porto Rico, Deaths from, 586
Treatment by Old Anti-tetanic Serum of Pasteur Inst.; Recovery, **596**
Titles of Unnoticed Papers, 599–600
Urethritis, due to a Pasteurella: Alexandria, **578**
Wassermann Test in India, **593**

**MYCOLOGY, TROPICAL,** 758–69
DISEASES
Actinomycoses
Experimental
Immunity Factors, **759** *bis*
Studies on, **759**
Immunity to Acquired and Natural Infection, Anatomical Basis and Cellular Factor in, **759**
Facial, **760**
Incidence
Cuba, 760
Greece, **761**
Serological Reactions in, **760**
Black Tongue: Malaya: in European, 759
Blastomycosis(es)
Chromoblastomycosis and Guiteras's Disease, "Chappa," **764**
Incidence
Cuba, **764**
Sumatra, **762**
Due to Yeast-like Fungi, **769**
Generalized, **763**
Human
of Mouth and Naso-pharynx in Latin America, **763**
Organisms Causing, Seven Strains of, Comparison of, **764**
Incidence
Brazil and Latin South America, **762, 763, 764**
U.S.A., 763
Systemic: Fatal Case, **763**
Bronchomoniliasis, in Porto Rico, **769**

**Mycology, Tropical**—*cont.*
DISEASES—*cont.*
Bronchomycosis, **761** *quat.*
Forms Simulating Pulmonary Tuberculosis, **761**
*Penicillium bertai* n. sp., Causing in Man, **761**
Dermal Nodule due to Thorn Infected with a Fungus, **769**
Gandy-Gamma Nodules, **768**
Granuloma, Coccidioidal: California, **765**
Treatment with Antimony and Pot. Tartrate, **765**
Moniliasis, Pulmonary, *see* Bronchomycosis, *supra*
Mycosis of Spleen, **765** *bis*, **766** *ter*, **767** *bis*, **768** *ter*, **769**
Prevalent in Brazil, 758
Singapore Ear, **759**
FUNGI
and Fungous Diseases, **758**
Pathogenic, Culture Medium for, **758**
Studies on, **758**
Title of Unnoticed Paper, 769

**OPHTHALMOLOGY, TROPICAL,** 504–10
Cataract
Double, after Thyroidectomy, **506**
Etiology, **506,** 509
Extraction
Conjunctival Bridge in, **507**
Intracapsular, without Iridectomy, **507**
Incidence
China: Yunnan, 416
Egypt, 510
Federated Malay States: Ipoh, 510
Juvenile, Resorption of, Spontaneous, **506**
Malaria as Cause of, **506**
Conjunctiva, Affections of, *see also* Trachoma, *infra*
Conjunctivitis
in Federated Malay States: Ipoh, 510
Phlyctenular, **504**
Pterygium, Recurrent, **504**
Schistosomiasis of, 509
Cornea, Affections of
Keratomalacia in Children; Kuala Lumpur, 510
Ulcer, **506** *bis*
Cysticercosis
Orbital, 509
Subretinal, **508**
Eyelids, Amyloid Degeneration of, **504**
Eye-Structures, Effect on, of Ultra-Violet Light, **509**
Glaucoma
in Federated Malay States: Ipoh, 510
Nutrition of Lens and Vitreous, **507**
Treatment by Hypertonic Saline Solution, **507,** 509
Lens, Affections of, *see* Cataract, *supra*
Lysozyme, Antibacterial Body, Present in Great Concentration in Tears; Relation of, to Infection of Human Eye, **508**
Ophthalmia
Catarrhal: Seasonal Incidence; Kuala Lumpur, 510

**Ophthalmology, Tropical**—*cont.*
Ophthalmia—*cont.*
Gonococcal, Acute
Egypt: Treatment, 509, 510
F.M.S.: Ipoh, 510
Pannus, Trachomatous
Treatment
New Method, **505**
Triphal, **505**
Report of
Ophthal. Dept., General Hosp., Kuala Lumpur, **510**
Ipoh Hosp. Ophthal. Dept. (1927), **510**
Ophthal. Mem. Lab., Giza (1927), **509**
Ophthal. Sect. Govt. of Egypt (1926), **509**
Tears, Lysozyme Content of, **508**
Trachoma
Incidence
Bolivia: Severe Epidemic, 1916, in School Children, **505**
China: Yunnan, 416
Dutch East Indies
Java: Serang, in School Children, **505**
Egypt, 509
Federated Malay States; Ipoh, in Chinese, 510
Treatment by
Chaulmoogra Oil, **505,** 509
Triphal (a Gold Preparation), **505**
and Tuberculosis, Association between, **505**
Ultra-Violet Light, Effect of, on the Eye-Structures, **509**

**OROYA FEVER AND VERRUGA PERUANA,** 54–5, 463–4, 849–50
*Bartonella bacilliformis* associated with
Cultivation of, **850**
OROYA FEVER
Etiology, **54, 463, 849**
Experimental
Chemotherapy in, **463**
in Monkeys, Influence on, of Malarial Infection, Splenectomy or both, **54**
Insect Vectors of, **849**
VERRUGA PERUANA
Haematology of, **55**
Investigations into, **463**
Verruga Peruana, **55, 850**

**Pappataci Fever,** *see* **DENGUE AND PAPPATACI FEVERS**

**PELLAGRA,** 356–9, 959–63
Associated with Organic Disease of the Gastro-Intestinal Tract, **960**
Blood Conditions in
Blood Plasma and Cerebro-Spinal Fluid in, pH of, **358**
Morphological Blood Changes in, **962**
Red Cell Sedimentation in, Rate of, **963**
Etiology, 961, 963
Experimental, in Rats. Dermatitis of, Etiology of, **359**
Following Voluntary Reduction of Diet, **358**

**Pellagra**—*cont.*
Food in relation to, **358, 959,** 960, 962
Incidence
Age, 961, 962
Geographical
Hungary, South, **357**
Malaya, **959**
U.S.A., 358
California, **960**
South Carolina
Endemic in Cotton-Mill Villages, **356, 963**
Southern States, 356, 960 *bis*, **961**
U.S.S.R.
Abkhasia, **962**
Georgia, **962**
Endemic, **357**
Race, 959
Season, 961
Sex, 960, 961, 962
and Keratomalacia in Malaya, **959**
Maize in relation to, 962, **963**
Pellagra, **356**
Titles of Unnoticed Papers, 359
Treatment, Germinated Maize in, **953**
Vitamins in relation to, 959, 960, 961

**PLAGUE,** 91–101, 633–42
Antiplague Bacteriophage in Paris, **638**
Vaccine Prepared with, **638**
Bacteriology
*Pasteurella pestis*
Colonies of, Atypical Forms of, **642**
Cultures of *B. pseudotuberculosis* of Rodents in relation to, **99**
Extracts of, Immunization by, of White Rats, **638** *bis*
Growth and Virulence of, Influence on, of Frost and Thaw, **637**
and *Past. pseudotuberculosis rodentium*, Relationship between, **639, 640** *bis*, **641**
Two Variations of, in Relation to Glycerin, **637**
Vitality of, Influence on, of Fixation and Staining, **638**
Black Death, in Danish Folklore and Tradition, **285**
Book on, Reviewed, **518**
EXPERIMENTAL
Antiplague Bacteriophage, Sensitization and Immunization with, **639**
First Plague Congress, U.S.S.R., 1927, Report of, **92**
Hibernation Experiments in, **98**
HUMAN
BUBONIC
Notes for Study of, **98**
Rats Transmitting, 98 *bis*
Incidence, all forms, or more than one form
Geographical
Aden: Epidemic (1928), **635**
Algeria: Oran, **633**
Argentina, **98,** 635
China, North, Spread of, Risks of, **635**

**Plague**—*cont.*
HUMAN—*cont.*
Incidence—*cont.*
Geographical—*cont.*
India, Yearly Variations in, in relation to Climate: Forecasting Epidemics, **633**
Kenya, 407
Nairobi, **99**
Mongolia; Epidemic Aug.–Nov., 1928, **634**
Nigeria, 1927, **91**
Porto Rico, Epidemic, 1921, Epizootic Survey of, **91**
Sweden, 1927, **91**
Transbaikalia, 636
Tripolitania, 633
U.S.A.
California, Del Monte, **634**
U.S.S.R., South-East, **92**
Seasonal, 633
PNEUMONIC OR PULMONARY
Incidence
Dutch East Indies: Java, **634**
Primary, Haemoculture in, **636**
Treatment, **99**
Prophylaxis
Deratization, 635
Gas and Chloropicrin, Effect of, on Susliks, **641** *bis*
Immunization of White Rats, **638** *bis*
Nairobi Plague Prophylactic, Bacteriology of, **99**
Rat-proofing of Buildings, **101, 635**
Transbaikalian Methods, **636**
Vaccine for, of Anti-Plague Bacteriophage, **638**
RODENT
and a Plague-like Septicaemia of Rats, **100** *ter*
in Spermophiles, Spontaneous: Transbaikalia, **641**
Rodents and Fleas, Geographical Distribution of, in relation to the Disease, **95**
Hibernating, as Sources of Infection, 98
Studies in, **636**
Titles of Unnoticed Papers, 101, 642
Transmission by
Fleas of the Genus *Xenopsylla*, **98**
Gerbilles, **641** *bis*
Ground-squirrels, 634
Marmots, 635
Rats, 98 *bis*
Healthy, but Carriers, 635
Susliks, Effect on them of Gas, and of Chloropicrin, **641** *bis*
Tarabagans, 635, 636

**Pneumonia and other Pneumococcic Infections**
Incidence
Belgian Congo, 410, 411
Kenya, 407
Treatment by Manganese, Remarks on, **587**

**RABIES,** 215–26, 725–39
ANIMAL, **225**
Antirabic Sera, Natural, as also Antivenemous, **739**

**Rabies**—*cont.*
CANINE
Experimental, *see infra*
Haemorrhagic Splenomegaly in, **220**
Incidence
Haiti, Port-au-Prince, **736**
Morocco, **737**
Paralytic Accident during Treatment, **735**
Pneumogastric Nerve as Infective in, **218**
Prophylaxis, **225** *bis*
Recurrent, **727**
in Cocks, **736** *bis*
EQUINE, **736**
EXPERIMENTAL
Canine, Effect in, of Fermi's Vaccine, **737**
with Fixed Virus, Glycaemia in, **219**
in Rabbits, Corpuscular Ratios in, Changes in, **220**
HUMAN
in Antiquity, **287**
Blood Picture in Child, Untreated, **220**
Carriers of, Possible Existence of, **225**
Case Reports, **728**
Cure-possibility Discussed, **219**
Diagnosis, **728**
Complement Deviation of No Value in, **221**
Pathological Changes in, **220**
Incidence in Animals and Man, *see also* CANINE *supra*
Brazil, 222
Canada (1925–6–7–8), **226**
China, **735**
Equatorial Africa, 725
France, 222
Germany, **734**
Hungary, **225**
India, 736
Assam, 222
Coonoor, **735**
Kasauli, 223, **735**
Malaya, Outbreak (1929), at Negri Sembilan, **736**
Siam, 736
South Africa, from Meerkat Bites, **725**
U.S.A., 223
Southern States, **739**
Infection of, Influence of, on *Spirochaeta duttoni* in Brains of Rats Immune to Recurrent Fever, **738**
Instructional Articles on (U.S.A.), **226**
Miscellaneous References, **225**
Myocardial Lesions in, **220**
Negri Bodies in, **726**
in Brains of Treated and Untreated Cases, **726**
Staining Method for Rapid Demonstration of, **728**
Negri Body-like Corpuscles in Nerve-fibres in, **225**
Parasite of, Two Phases of, in Nature, **225**
Pasteur Institutes, Statistics of, **222, 734**
Pathology, **220, 728**
Prophylaxis
Immunization of Animals, **732, 733**
at Kedah, Malaya, **226**
Local, **222**

**Rabies**—*cont.*
Prophylaxis—*cont.*
Non-Specific, **221**
Vaccination
of Dogs
Japanese Method, **737** *bis*
Morocco, **737**
Value in, of the Haemato-Encephalic Barrier, **728**
Legislative, Suggested for Southern U.S.A., **739**
Results in Hungary, **225**
Reviews of Recent Articles, **215, 725**
Saliva from Fistula in Wharton's Duct, Infectivity of, Experiments on, **217**
of Patient under Treatment, Infectivity of, **225**
Salivary Glands in, Infectivity of, Prior to Appearance of Symptoms, **728**
Statistics, **222, 734**
Symptoms, **727**
Transmission, Experimental, Site for Puncture, **738**
Treatment, **222**
Accidents during
Paralytic, **223** *ter*, **735**
A Correction, **224**
Dosage and Heat as Causing, **224**
Actual Position of, **226**
Anti-rabic Sera for, Various, Rabicidal and Immunizing Power of, Compared, **729**
Various Methods, **221, 730**
Destruction of Virus at Seat of Inoculation, **734**
Fresh Virus, Experiments with, **733**
Harris's: U.S.A., 728
Statistics of, 223
Nytriosaure, **734**
Sera of Guineapigs after Treatment with Phenol-Glycerinated Vaccine, Fixed Virus Inactivated by, **731**
Splenectomy, Effects of, **729**
Stuart and Krikorian's, **732**
Utilization of Immunizing Properties of Nucleo-Proteids from Rabies Brains, **221**
Vaccination with
Carbolized Vaccines, **222**
Etherized Vaccine, **219** *ter*, **221**, 223, **225**
by Meningeal Route, **222**
Fermi's Vaccine, **221**
Formol-Vaccine, **221, 222**
Glycerine Vaccine, Comparison of Results with those of Carbolized, **731**
Högyes', Phillips', and Semples' Vaccines, Statistics of Results, **734**
Ustupny's Method, **738**
Vaccines for, Immunizing Potency of Various: Single Dosage with, **737**
Virus Attenuated with Gastric Juice: Originator of Method, **226**

**Rabies**—*cont.*
Treatment—*cont.*
Various Methods—*cont.*
Vaccination—*cont.*
Vaccines for—*cont.*
Production of, **730, 731**
Phenol as best Antiseptic during, **731**
Ponomareff and Soloview's Method, **732**
Remlinger's Ether, Value of, **737**
Short Intensive use of, Risks from, **733**
Trephining Instrument for Rabbits, **738**
and Vaccinia, Cross-Immunity between, Negatived, **219**
Virus, **216** *sqq.*
Action on, of Ultra-Violet Rays, **738**
Dispersion of, in Peripheral Nerves of Rabbits after Intracerebral Inoculation, Rate of, **218**
Hypervirulence of Strain obtained from Cow, **726**
and Indian Ink Injections, **729**
Nature of, **215**
Transmission of
Along Nerve Trunks, **217, 225,** *cf.* **218**
Through Placenta, **225**
as Used in Treatment, **226**
Fixed
Budapest Strain, Loss of Virulence of, Time taken, **221**
Characteristics of, **216** *bis*
Effect on, of 1 per cent. Carbolic Acid, **219**
Estimation of, **217**
French and Indian, Action on, of Ether, **219** *bis*
Inoculation with, Glycaemia after, **219**
Negri Bodies in, **217**
and the "Neuro-infections mortelles auto-sterilisables," **217**
Virulence of, Duration of, and Loss of, **727** *bis*
Fixed and Live, in Brains of those Dying During or After Treatment by Live Virus, **224**
Fixed and Street, Relative Aptitude of for Transmission along Nerves, **218**
Neurotropic, **727**
Plurality of Strains
as Affecting Treatment, **725**
Not proven, **725**
Street
Characteristics of, 216
Spread of, in the Central Nervous System, **218**
Viruses, Filterable, Nature of; Transition from Live to Dead, **215**
Wounds of, Treatment of, **226**

**RAT-BITE FEVER,** 135–9, 674–6
Biology, Clinic Observations and Etiology, **135**
Experimental
Blood and Haematopoietic Organs in, Structure as affected by, **137**
Study of, **136**
Treatment, Aetiotropic, **675**
in White Mouse, Chemotherapeutic Trials in, **138**

**Rat-Bite Fever**—*cont.*
Grave Case, **675**
Incidence
Age, **138,** 674, 675
Geographical
Brazil, Rare, **138**
Canada: Montreal, **138**
China: Yunnan, 416
Dutch East Indies, 674
Hong Kong, **675**
India, **139**
Laboratory-contracted, **139**
Literature on, Critical Review of, **136**
Pathogenesis of, **674**
Serodiagnosis of, **138**
Sodoku Treatment in Paresis: Report on 72 Cases, **675**
Titles of Unnoticed Papers, 676
Treatment by Neojacol, 675

**RELAPSING FEVER AND OTHER SPIROCHAETOSES,** 113–28, 651–68
Antibodies as Protective Factors in, **420**
Cases with Biological Peculiarities, **115**
Exanthem in, **114**
Experimental
Action on, of Gold Compounds, **653**
Immunization in, **661** *bis*
in Rodents, 663
Special Neurotropism of *S. duttoni*, **663**
Sterilization of, by Salvarsan, **662**
Treatment with Sulphoxylsalvarsan, **119**
with *S. duttoni*, Immunity, and Diversity of Strains in, **115**
Historical Reference to, **285**
Immunity in, Nature of, **420**
Incidence
Abyssinia, **114**
Belgian Congo: Kasai Region, 411
Central Asia, and its Pathogenic Agent, **651**
Bukhara: Fergan, **651**
Samarkand, Province of, **651**
China, 117
Colombia, Western, 585
Gallipoli, in Bulgarian Troops, 114
Morocco, 115, **116, 652** *ter*
New Spirochaete from, **657**
Nigeria: Kano, 653
Spain, **651, 653**
Urundi, **652**
Laboratory Infection Cases, **116** *bis*
One with Two Strains Simultaneously, **657**
Meningismus in, **653**
Moroccan, Note on, **652** *ter*
Pathology, Experimental Study of, **653**
Prophylaxis
Inoculation, Experiments on, **656** *bis*
Other Spirochaetoses
Spleen in, Significance of, **126**
Bronchospirochaetosis, **121, 122** *bis*
Incidence
Argentine
Santiago del Estero, 121, 122
Dutch East Indies
Bali Island, **665**
Moluccas, **122**

**Relapsing Fever**—*cont.*
OTHER SPIROCHAETOSES—*cont.*
BRONCHOSPIROCHAETOSIS—*cont.*
Incidence—*cont.*
Greece, 666
Morocco: Epidemic, **120**
Palestine, 122
Studies in, **666** *quat.*
Ecto-Mesodermic, **126**
Rieckenberg-Brussin Reaction in Human, **127**
Technique of, **125**
Spanish Form
in Mice, Chemotherapeutic Study of, **119**
Spirochaete of, in Morocco, **116**
Transmission of, Rôle in, of *Pediculus vestimenti*, **659**
Spirochaetes (Spirilla, Spirochaeta, Treponema) Associated with
Book on, Reviewed, **426**
Comparative Studies of, by Method of Agglutination, **665**
Culture of, **120**
Culture Media for, **123** *bis*
Dakar Strains, Various, Identity of, **116** *bis*
Experimental Adaptation of, to Ornithodorus other than those Naturally Transmitting, **658**
Fowl, First Attempts to Adapt to *Ornithodorus* sp., **118, 668**
Intestinal, Question of, **667**
Kept in Labs.; Necessity of Identification and Control of, **659**
Letter from the Discoverer of, Otto Obermeier, **285**
Measurement of, **128**
Mouth, Portuguese India, **123**
Neurotropisms of, **113** *ter*, **114**
Influence on, of External Conditions, **663**
N. sp. from Moroccan Case, **657**
Peculiar Structure in Some, **128**
Review of the, **127**
Settling in the Nerve Tissue, Process of, Rôle of Injury in, **663**
of Spanish Form, in Morocco, **116**
Stain for, Ziehl's Fuchsin, **128**
Staining Properties of, Comparative, **128**
Strains kept at the Pasteur Inst., Tunis, List of, 659–60
and Treponemata from a Case of Granuloma Venereum, **667**
and Trypanosomes
Action on, of Free Arsenic, **127**
Mixed Infection with, **126** *bis*
Species referred to by Names
*crocidurae* and *hispanica*
Experimental Studies of, **664**
*duttoni*
in Mouse's Brain, **662**
Neurotropism of, **663**
Therapeutic Action on, of Antimony, **119**
*gallinarum*, Virulence of, Revival of, by Inoculations of *Argas persicus* into Chicken and Fowl Embryos, **118**
*hispanicum*, **664**
Culture of, **665**

**Relapsing Fever**—*cont.*
Spirochaetes—*cont.*
Species referred to by Names—*cont.*
*recurrentis*
Morphological Variability of, **660**
Relapse Phenomena of, **117**
Resisting Power of, to Temperature, Light, and Darkness, **665**
*sogdianum* n. sp., Pathogenic for Guineapigs, **118**
Transmitted by *O. papillipes*, 668
Treponemata from and from Granuloma Venereum, **667**
Titles of Unnoticed Papers, 128, 668
Treatment
Experimental Study of, **653**
Various Methods
Spanish, **653**
Stovarsol, **115**
Used in G.P.,I. Observations on, **654, 656**

**REVIEWS OF BOOKS, AND NOTICES,** 75–82, 166–8, 345–8, 425–38, 511–18, 601–6, 691–3, 782–4, 867–8, 952–8
Actas y trabajos do Segundo Congreso Nacional de Medicina, Buenos Aires (1 al 8 de Oct. 1922) Tomo VI. Bibliografia de Hidatidosis (Tumburus compiler), 605–6
African Conference of the Yellow Fever (Dakar—April, 1928 (General Government of French West Africa), 602–4
Annual Report of the Calcutta School of Tropical Medicine, Institute of Hygiene, and Carmichael Hospital for Tropical Diseases (Acton), 438
Anthelmintics and their Uses in Medical and Veterinary Practice (Chopra & Chandler), 512
Army Health in India. Hygiene and Pathology (Mackenzie, foreword by Fell), 868
Beiträge zur medizinischen Entomologie und zur Malaria-Epidemiologie des unteren Wolgagebiets, 81
Boletìn dèl Instituto de Clìnica - Quirùrgica [Argentina], Vol. 4, 78–80
Catalogue of Lewis's Medical and Scientific Circulating Library. Part I. Authors and Titles. Part II. Classified Index of Subjects with Names of Authors who have written upon them. New edition, Revised to end of 1927, 958
Découverte, La, de Laveran. Constantine 6 Novembre 1880 (Sergent (Edm. & Et.) & Parrot), 693
Diagnostics, The, and Treatment of Tropical Diseases (Stitt), 515–17
Diseases, The, of China, including Formosa and Korea (Maxwell), 511
Epidemiologie und Tropenkrankheiten in China (Nauck), 167–8

**Reviews, &c.**—*cont.*
Epidemiology, The, of Leprosy in Australia (Cook), 347
Esculape chez les Artistes (Cabanès), 346
Exotische Krankheiten. Ein Lehrbuch für die Praxis (Mayer), 783
Filariosen des Menschen (Fülleborn), 513
Four Centuries of Medical History in Canada and a Sketch of the Medical History of Newfoundland (Heagerty), 345
Gelbe Fieber, Das (Hoffmann), 952
Grande Page, Une, de l'Histoire de la Médecine. La Découverte de la Transmission du Paludisme par les Moustiques (Ross: préf. et introd. Broquet), 427
Graphic Guide, A, to Elementary Surgery (Nageli, *trs.* Snowman, *introd.* Garré), 782
Handbook, A, on Diabetes Mellitus and its Modern Treatment (Bose), 426
Handbuch der Tropenkrankheiten (Vol. on Helminthology) (Mense), 867
Herz und Kreislauf bei der Beri beri-Krankheit (Aalsmeer & Wenckebach), 517
Hydatid Disease. Its Pathology, Diagnosis and Treatment (Dew), 691–2
Indian Therapeutics. Dealing with the Comparative Diagnosis, Ayurvedic Materia Medica, Dietetics, etc., for Students and Practitioners (Sandu), 82
Insect Nutrition and Metabolism. A Summary of the Literature (Uvarov, pref. Hemming), 433–4
Laboratory Manual of the Division of Bacteriology (ed. Lim), 867–8
Leerboek der Tropische Geneeskunde (de Langen & Lichtenstein), 430
Leitfaden zur Untersuchung der tierischen Parasiten des Menschen und der Haustiere (ed. Reichenow), 692–3
Letters from Rome on Certain Discoveries connected with Malaria. Charles & Calandruccio etc. (ed. Ross), 956
Ligue Antimalarienne Hellénique (1905–28) (Savas & Cardamatis), 428–9
Lutte, La, contre le Paludisme en Corse (Sautet), 166–7
Malay Poisons and Charm Cures (Gimlette), 168
Manson's Tropical Diseases. A Manual of the Diseases of Warm Climates (ed. Manson-Bahr), 429–30
Manual, A, of External Parasites (Ewing), 957
Manual, A, of Helminthology, Medical and Veterinary (Baylis), 432–3

**Reviews, &c.**—*cont.*
Mass Diagnosis, The, of Hookworm Infection (Lane), 605
Medical Museum, The. Modern Developments, Organisation and Technical Methods based on a New System of Visual Teaching. An Amplification of a Thesis read for the Degree of M.D., Cambridge (Daukes), 953
Mosquitoes, The, of the Americas (Dyar), 514–15
On the Dysenteries of India. With a Chapter on Secondary Streptococcal Infections and Sprue (Acton), 77–8
Perniciosità, La, nella Malaria (Marchiafava), 80–1
Physiology and Biochemistry of Bacteria. Vol. I. Growth Phases: Composition and Biophysical Chemistry of Bacteria and their Environment: and Energetics (Buchanan), 82
Poisonous Terrestrial Snakes, The, of our British Indian Dominions (including Ceylon) and how to Recognize them. With Symptoms of Snake Poisoning and Treatment 4th ed. (Wall), 348
Primo Censo de la Lepra (Urena), 425–6
Protozoology. A Manual for Medical Men (Thomson & Robertson), 435–6
Recent Advances in Tropical Medicine (Rogers), 963
Researches in Polynesia and Melanesia. Parts V-VII (Relating to Human Diseases and Welfare), (Buxton), 436–8
Reports 1927–28, North Manchurian Plague Prevention Service (Wu Lien Teh), 518
Rockefeller Foundation. A Review for 1928 (Vincent), 954–6
Spirochäten, Die (Nachtrag) (Zuelzer), 426–7
Studies on Malaria (Ross), 166
Subsidios ao estudo da "Framboesia Tropica" (Araujo), 345
Surgery in the Tropics (Connor), 430–2
Transactions of the 7th Congress of the Far Eastern Association of Tropical Medicine (ed. Cunningham), 517–18
Travaux Pratiques de Parasitologie (Brumpt), 434–5
Tropical Nursing. A Handbook for Nurses and others going Abroad (Gregg), 606
Tsé-Tsés, Les. Tome Premier. Généralités. Anatomie. Systématique Réproduction. Gîtes à Pupes. Ennemis Prédateurs et Parasitiques (Hegh), 601–2

**Reviews, &c.**—*cont.*
Twenty-Five Years of American Medical Activity on the Isthmus of Panama 1904–1929. A Triumph of Preventive Medicine (Chamberlain), 783–4
Use, The, of Standard Treatments in the Campaign against Disease in the Tropics, (Leitch, introd. Low), 957–8
Züchtung, Die, der parasitischen Protozoen (Nöller), 757

**SLEEPING SICKNESS,** 185–214, 695–724
Chemotherapy
Antimony Therapy
Liver-Necrosis, Incipient, due to, Detection of, by the van den Bergh Reaction, **711**
Bayer 205 and Naganol, Antitrypanosomal Activities of, Comparison of, **213**
Chemotherapeutic Curative Processes, Mechanism of, **203**
in Experimental *T. brucei* Infection, **710**
Germanin
Action of
Antimutative, Question of, **711**
Possibility of Improving, **709**
Trypanocidal Action of Normal Human Serum, **716**
Trypanocides, Comparative Value of those employed in the Cuanza Sanitary Zone, **709**
GLOSSINA
Book on, Reviewed, **601**
*palpalis*, Bite of, Pathology of, **703**
Report on, of 2nd International Conference of the League of Nations Health Organization, **185**
Titles of Unnoticed Papers, 724
Trypanocidal Substance Content of Children's Blood, **213**
Trypanocidal Test, Standardization of, with Arsphenamine, **212**
Trypanolysis, *in vivo*, with Reference to Zones of Inhibition, Relapse Phenomena, and Immunological Specificity, **719**
TRYPANOSOMES
Anaphylaxis by, **212**
Developing from a Flagellate Parasite of *Triatoma rubrofasciata* in Lab. Animals, **718**
Developmental Cycle of, in the Mammalian Host, **712**
Infections with
Resistance to, Influence on, of Starvation and Lack of Vitamins, **714**
Rat-Resistance, **715**
Relapse Strains, Rieckenberg-Phenomenon in, **208**
Reservoirs of, Wild Game as, Estimating the Adhesion Phenomenon in, Value of, **701**

**Sleeping Sickness**—*cont.*
TRYPANOSOMES—*cont.*
Species referred to, by Names
*brucei*, Experimental Infection with, Bacteriotherapy and Chemotherapy in, **710**
*equinum*, Infection with, Non-Occurrence in, of Zone Phenomenon, in Curative Action of Drugs and Normal Human Serum in, **720**
*evansi*, and *equiperdum*, Action on, of Bayer 205, **213**
*gambiense*, Infection with, Clinical Notes on, 577
and *rhodesiense*, Immunological Studies of, **204**
*grayi*, Effect on, of Goat's Blood, **212**
*rhodesiense*, Inoculations of Animals from Cases of Infecton with, **206**
Treated with Immune Serum, Do they act as Antigen ? **720**
and Treponemes, Mixed Infection with, Causes Influencing Development of, **723**
TRYPANOSOMIASES
ANIMAL
Blood Sugar in, **721, 722** *bis*
Curative Experiments in, with Non-Specific Means, **709**
Incidence
Tanganyika Territory
Ikoma District, **206**
Oedemas in, Etiology of, **717**
Antibodies, Natural, and Placental Permeability, **208**
EXPERIMENTAL
Acute, Hypoglycaemia in, Death from, **209**
Effect of, on Haemo-Encephalic Barrier, **207**
Immunity in, **721**
Immunological Studies on, **212**
Inoculations of Animals from Cases of *T. rhodesiense* Infection, **206**
in Rats, Blood of, Lactic Acid Content of, **718**
Relapsing Fever as Influencing Development of Certain Forms, **723**
Treatment by
Atoxyl and Tetanic Antitoxin, **202**
Etharsanol and Proparsanol
Biologic Study of, **198**
Therapeutic Value of, **199**
Stovarsol Sodium, **202**
with *T. brucei* Infection, Bacteriotherapy and Chemotherapy in, **710**
HUMAN, AFRICAN
Chemotherapeutic Discoveries affecting Prophylaxis and Treatment, 196–7
Chronicity of, Question of, **704**
Diagnosis, Adhesion Phenomenon as Aid to, **701**
Formol-Gel Test in, **211**

**Sleeping Sickness**—*cont.*
TRYPANOSOMIASES—*cont.*
HUMAN, AFRICAN—*cont.*
Incidence
Geographical
Angola, Cuanza Sanitary Zone, **709**
Belgian Congo (1927), **698**
Campaign against, **191** *bis*, **193, 197**
Kasai Mines, Medical Services and, 409, 410, 411
Fernando Po, 703
French Africa, **699**
French Cameroons: Fight against, **190**
Mozambique, Work in, of Medical Mission, **189**
Sudan, Report on (1926–7), **700**
Tanganyika Territory, **206, 695, 696, 697**
Togoland, Epidemic Focus in, **190**
Uganda, Report on, for 1927, **187**
in Mother and Suckling, **702**
Incubation Period in, **703**
Initial Lesion of, **703**
Prophylaxis
Bayer 205
Critical Review, **196**
in Itinerant Practice, **193**
Belgian Campaign, **191** *bis*, **193, 197**
French Campaign, **190, 699**
Rhodesian, Dermatitis associated with Bayer 205 Treatment of, **709**
Second Stage of, Takata-Ara Reaction in, **722**
Treatment, and Prophylaxis (*q.v.*) of, Position of, **196**
Various Methods
Bayer 205
Critical Review, **196**
Reports on Cases so treated, **708, 709**
Fourneau 269 and 417, **198**
Germanin, *see* Bayer 205, *supra*
Tryparsamide, **724**
Experiences with, **707**
Tryponarsyl, Arsenic Resistance in, **708**
HUMAN, AMERICAN OR CHAGAS'S DISEASE
Incidence
Argentina, **203, 704** *bis*, **705**
Diagnosis of, **704**
Mexico: Vera Cruz, Probable Vector, **700**
U.S.A: New York, **705**
Parietal Endocarditis in, **203**
Trypanotoxins, **210**
Wild Game Trypanosome Reservoirs, Estimating of, Value in, of the Adhesion Phenomenon, **701**

**Smallpox**
Incidence
Belgian Congo: Kasai Mines, 409
China: Yunnan, 416

**Sodoku**, *see* **RAT-BITE FEVER**

**SPRUE**, 490–502
Anaemias of, 491, **491, 492**
Treatment by Liver Extract, **494, 495, 496, 497** *bis*
Analytical Study of 150 Cases, **490**
Animal Experiments and, **499**
Bacteriology of, **497**
Aerobic Flora of Stools of, **502**
*Bacillus*, Unrecognized, Isolated from, **501**
Blood in
Agglutinins in, for Organisms of the Colon-Typhoid-Dysentery Group, **502**
Changes in, **492**
Book on, Reviewed, **77**
Bowel Disease, Association of, with Vitamin C Deficiency, **499**
Diagnosis, Differential, of, and of Pernicious Anaemia, 495, *see also* **492**
Diarrhoea, Grave, Resembling, **491**
Immunological Investigations on, **502** *bis*
Incidence
Belgian Congo: Katanga, **493**
China, 495
Countries from which London-treated Cases came, 490
Cuba, 491
Germany: Indigenous, **494**
Mexico, 491
Nigeria, 578
Porto Rico, 492, **493, 502** *bis*
U.S.S.R., **490**
*Monilia psilosis*, Biology of, in Relation to, **502**
Morbid Anatomy of, **500**
Organs after Death from, Microscopical Changes in, **501**
Recent Literature on, 503
Relation to, of Condition of Gastro-Intestinal Tract in Porto Rico, **493**
Study of, **491**

**Tsutsugamushi Disease**, *see* **JAPANESE RIVER FEVER**

**Tuberculosis**
Bronchomycoses Simulating, **761**
Dengue and, **836, 837**
Incidence
Belgian Congo: Kasai Mines, 410, 411
China: Yunnan, 416
Dutch East Indies, 415
Espiritu Santo, 416
Kenya, 407
Porto Rico: Death-rate, 586

**Typhus Fever, Tropical**
Etiology of, **50, 848**
Fever Resembling
Australia, **52**
Elsewhere, **461, 462**
Incidence
Federated Malay States, **848**
Indo-China, **848**
Mexico: Etiology, **50**
Rakka, **461**
Tonkin, **589**

**Typhus Fever, Tropical**—*cont.*
and Pseudo-Typhus in Indo-China, **848**
Tick-, and other Sporadic Fevers of the Typhus Group, **49**
India, History of Tick-Bites, **846**

**UNCLASSED FEVERS,** 47–52, 457–63, 842–9
Diagnostic Difficulties in the Tropics, **47**
Eruptive or Exanthematic
Classification of, 844
Distinct from Typhus, **460**
and Endemic Typhus, **457** *bis*
Identity of, **842** *bis*, **843,** 844
of the Mediterranean Coast, New Work on, **49**
Observations and Researches on, **458** *bis* **459** *bis*
Transmission Experiments with Lice, **460**
Febrile Diseases of the Baluchistan Plateau, **47**
"*Fièvre boutonneuse*" of Tunis and Eruptive Fevers of Marseilles and Italy, **842** *bis*, **843, 844** *bis*
Five-Day, Clinical and Experimental Investigations of, **463**
Hone's Endemic Typhus-like Disease: Death during Convalescence, **461**
Pyrexia of Seven Days' Duration; Origin Undetermined; Malaya, **845**
Septicaemia due to *Micrococcus tetragenus*, **52**
Short Fevers in India, **846, 847**
Tick Fever (?) from Poona: Two Cases, **462**
Typhus-like (? Col. Megaw's Tick-Typhus), **462**

**UNDULANT FEVERS,** 56–74, 439–49, 825–32
*ABORTUS* Infection
Bovine, Contagious, Prevalence of, in Denmark, 65
Clinical Analysis of 125 Cases, **827**
in Man, **60, 63, 442** *bis*, **443, 447** *bis*, **826, 827, 828**
Agglutination Test Applied to more than 10,000 Human Sera: Results, **70**
as Cause of Septic Diseases, **61**
Clinical Aspects of, **60**
Incidence, *see under* UNDULANT AND ABORTUS FEVERS
Titles of Unnoticed Papers, 74, 832
UNDULANT FEVER
Allergic and Agglutination Reactions in, **68**
Cases of Bovine and Caprine Origin, **57**
Chemotherapy of, **64**
and its Complications, **56**
Critical Study of, **825**
Cure by Vaccine Therapy, **64**
Diagnosis, **826**
Due to *Brucella abortus*: Studied in Denmark (*map*), **58**
Epidemiology of (*see also* Incidence, *infra*), **826**
Glycaemia in, **449**
Haemoclastic Reaction, the, in, **597**
Haemorrhagic Forms of, **446**

**Undulant Fevers**—*cont.*
UNDULANT FEVER—*cont.*
Immunization against, of
Goats, **68, 830** *bis*
White Rats, **445**
Incidence, *see under* UNDULANT AND ABORTUS FEVERS
Lung Infections, Specific, in, **65**
Prophylaxis
Vaccination with Cultures of *Br. abortus*, **444**
Pseudo-Tuberculosis Forms, of, **828**
Treatment
*Br. abortus* Protein, **829**
Melitine: Therapeutic Value of, **445**
Trypaflavin, 64
Intravenous: Cure, **446**
Vaccine: Cure, **64**
UNDULANT AND ABORTUS FEVERS
Bacteriology
*Br. abortus*
Agglutinating Action on, of Human Blood Serum, **831**
from Human Sources, Identification of, **441**
Is it Experimentally Pathogenic for Man? **72**
in Milk and Dairy Products, U.S.A., **58**
Thermal Death Point of, **448**
Pathogenicity of, for Man, **65,** 442 *bis*, 443
Studies on, Summarized, **73**
*abortus* and *melitensis*
Behaviour of, toward Gentian Violet, **73**
Differentiation of, by
Agglutinins, **447**
Chemical Substances, **448**
Inoculation of, in Goats, **67**
Pathogenicity of, for Man and for Monkey, **443, 829**
Relationship between, **71** *bis*
*Br. abortus-melitensis* Group or Species
Agar-Cultures of, Vitality of, **72**
Development of, Thermo-agglutination and, **69**
Metabolism of: Effect of Carbon Dioxide, **74**
*Br. melitensis* as Cause of Abortion in Cows, **832**
Incidence, **826**
Black Sea, East Coast of, **56**
Canada, 63
France, 828
Germany, 61
Holland, 62
Italy
Bologna, **444**
Lugo, Epidemic: Malaria Associated with, **57**
Sicily, 56, 832
Poland, 63
Sweden, **825**
Switzerland, 63, 827
U.S.A., 58, 60
Iowa: Epidemiology of, **439, 827**
Kentucky, 826
Michigan, 57
New York State, **56, 439**

**Undulant Fevers**—*cont.*
UNDULANT AND ABORTUS FEVERS—*cont.*
Incidence—*cont.*
U.S.A.—*cont.*
Pennsylvania, **60**
Utah, 57

**Weil's Disease,** *see, under* **LEPTOSPIROSES,** Spirochaetosis Icterohaemorrhagica

**YAWS AND SYPHILIS,** 169–80, 851–62
GANGOSA
in Western Colombia, 585
JUXTA-ARTICULAR NODULES
Incidence
Geographical: Somalia, **856**
Race
Japanese, **175**
Portuguese, **174**
Roumanian, **175**
in Syphilitics, **175**, **856**
LYMPHOGRANULOMA INGUINALE
Vulval and Anorectal Complications of, **861**
SYPHILIS
and Allied Tropical Diseases, Meinicke and Kahn Tests in, **180, 861**
American Origin, **204,** 289
Antenatal
Treatment of, **859**
Success of, 412
Course of, in Natives, D.E.I., **857**
Cutaneous, in Somalia, **858**
Epidemiology of, **588**
GENERAL PARALYSIS OF THE INSANE
Treatment by Relapsing Fever, **655, 656**
Incidence
Belgian Congo: Kasai Mines, 409, 410, 411
Brazil: Rio de Janeiro, **858**
Dutch East Indies, 414, 857
Java: Affections of Central Nervous System, due to, **861**
Haiti, 179
Madagascar, 861
Senegal: Dakar, 412
Somalia, **858**
Uganda, 412, 413, 859
Meinicke and Kahn Tests in, and in Allied Diseases in the Tropics, **180, 861**
Primitive, **177**
TABES DORSALIS
Treatment
Antenatal, Uganda, **859**
Success of, 412
Various Means
Bismuth, Side Effects of, **179**
Bismuth Salts, **852**
Tropical, Spinal Fluid in, **179**
Titles of Unnoticed Papers, 180, 862
TREPONEMATOSIS
Incidence
Haiti, 860
Syrian Desert, in a Bedouin, **177**
Treatment by Antimony and Potassium Tartrate, **860**

**Yaws and Syphilis**—*cont.*
YAWS
Book on, Reviewed, **345**
Case Reports, **174**
Clinical Development of, **170**
Experimental in Monkeys
Philippines, **172**
Wassermann Test in, Effect on, of Alcohol, **855**
Experiments in, Results of, **172**
Goundou (*q.v.*) and, **173**
Hand-affections due to, **174**
Incidence
Geographical
American Countries, Apparent Disappearance from these, **170**
Brazil, **169**
Cochin China, 170, 851
Colombia, Western, 585
Congo, Belgian, **854**
Kasai Mines, 409, 411
Katanga, 171
Portuguese, 856
Costa Rica, **852**
Cuba, 171
Gold Coast, 852
India, 171
Bengal: Rare, **593**
Kenya, 409
Nigeria: Benin City, 174
Oceania, 418
Philippine Islands, **856**
Rhodesia, Northern: Kasempa District, **852**
Somalia, **851**
Uganda, 852
Late, *Lichen spinulosus* a Manifestation of, **171**
Prophylaxis
Control Measures in Costa Rica, **852**
Employed in Belgian Congo, **854**
Salvarsan-Resistant Cases, Two, **854**
Treatment
Airol, **171**
Bismuth, New Organic Aromatic Compound of, for Intravenous Injection, **171**
Bismuth Salts in Africa, **852** *ter*
Myosalvarsan: Successful, **856**
"Neo I.C.I.," **854**
Various Drugs, **853**
in Belgian Congo: Results, **854**
Yaws Chancre, The, **851**
YAWS AND SYPHILIS
Incidence
in East Africa, 178
in Light of Recent Experimental Work, **175**
Treatment by Bismuth: Reports on Cases so Treated, **178**

**YELLOW FEVER,** 292–309, 643–50, 994–1008
*Aëdes aegypti,* Excreta of, Transmission by, of Virus of the Disease, **1001** *bis*
Male, Infection of, with Yellow Fever Virus, and Possible Transmission thereof to Man, **1001** *bis*

**Yellow Fever**—*cont.*
Blood Coagulation in, in Man, and in Experimental Monkeys, Modifications in: Value of, for Diagnosis, **1002** *quat*, **1004**
Books on, Reviewed, 602, 952
Can it Spread into all Stegomyia Countries? **650**
Cerebro-Spinal Fluid in, **299** *bis*
Clinical Records of, **300**
Clinical Study of, **995**
Complement in, Amount of, **1004**
Complement Fixation Test in, **1003**
Concerning, **1008**
Diagnosis
Bacteriological, **998**
Consideration of Symptoms of Import for, **996**
Differential, in Childhood, **298**
Early, Prophylactic, **995**
Modifications of Blood Coagulation as Important for, **1002** *quat*
Electrocardiogram in, **996**
Epidemiology of, Present State of our Knowledge of, **296**
Etiology, Studies of, **296**
Europe and, **1008**
Experimental: Brazil, **1008**
Difference of Virulence in Viruses Used, **646**
Intranuclear Inclusions in, **648** *bis*, **997** *bis*
in Monkeys: Blood Coagulation in, Modifications of, **1004**
Experimental Study of, An, **292**
Experiments on, at Oswaldo Cruz Institute, **1002**
First Stages of, Prophylactic Diagnosis and Symptoms in, **995**
Haematological Studies in, **649**
Human, Intranuclear Inclusions in, **998**
Immunological Relationship of, as Occurring in West Africa and in South America, **306**
Incidence
Age
Childhood, **646, 994**
Differential Diagnosis in, **298**
Geographical
America, South, **306, 999, 1000** *bis*
Belgian Congo
Boma, **645**
Lower Congo, Report on, **293**
Brazil, 646, 649
Rio de Janeiro, **994**
1928 Epidemic, **294** *ter*, 298–301, **301, 302** *sext*, 303, **305, 308**
Data on, **1008**
British West Africa
Accra, **645** *bis*, **646**
French West Africa
Dahomey and Ivory Coast, **644**
Senegal, Epidemic, 1927, **643** *bis*, **646**, 647, 649
Mexico [None recorded for Seven Years], **646**
Peru
Lambayeque Dept.: Epidemic of 1921, **994**

**Yellow Fever**—*cont.*
Incidence—*cont.*
Geographical—*cont.*
West Africa, **306**
Potential Danger from, to Europe, **1008**
Sex, **994**
Intramuscular Inclusions in
Experimental, **648** *bis*, **997** *bis*
Human, **998**
Jaundice with Yellow Vomit, Case of, Notes on, **997**
*Leptospira icteroides* and, **307, 308** *bis*
Search for, in African Cases of the Disease, **308**
Liver in
Cells of
Necrobiosis of, in, **301**
Oxychromatic Degeneration of, in the Brazilian Forms of the Disease, **301, 302** *sext*
Fatty Substances in, Amount of, **300**
Pathology of, **300**
Nervous System in, Changes in, **298**
Notes about, **295**
Pathogenesis of, Studies on, **296**
Prophylaxis
Anti-Yellow Fever Serum, Protective Qualities of, **307**
Campaigns against, Indices of Importance in, **995**
Insecticide-Sprinkling, **650**
Protection Tests, with Serum of Persons Recovered from, in the Western Hemisphere and West Africa, **647**
Vaccination, Hindle's and other Vaccines for, 304–5 & *n*.
Water Chlorination, 308
Research on
Holland, **1006**
Report on, **303, 305**
Summary of, **308**
Review of, **289**
Serum Reactions in, **1004** *bis*, **1005**
South American, Studies on, **999, 1000** *bis*
*Spirochaeta icterohaemorrhagiae* and *Leptospira icteroides*, Identity of, a Pathological-Anatomical Study of, **307**
Suprarenal Syndrome in, **299**
Suprarenals in, Lesions of, **299**
Susceptibility to, of *Macacus rhesus*, **1005**
Symptoms, Diagnostic Import of, **996**
Early, **995**
Titles of Unnoticed Papers, 309, 650, 1008
Transmission by
*Aëdes aegypti*, without Human Intervention, Possibility of, **1000, 1001**
Excreta of Infected Mosquitoes, **1001** *ter*
Mosquitoes other than *Aëdes aegypti*, **1005**
Transmission of, to *Macacus rhesus*, **303, 647, 1005**
Treatment
Anti-Yellow Fever Serum, Curative Properties of, **307**
Urine in, **649**
Some Elements of, **300**
Urinalysis in, **996**

**Yellow Fever**—*cont.*
Virus(es) of, **308**
Brazilian Strains, Transmission to Macaques, **647, 650, 1005**
Experiments with, on Monkeys, **647, 650,** 999, **1000** *bis*, **1005**
Infection with, of Male *Aëdes aegypti*. **1001** *bis*

**Yellow Fever**—*cont.*
Virus(es) of—*cont.*
Passage of, Through the Skin, **303,** 1005–6
Preservation of, **1007** *bis*
Strains of, in Use at the Lab., Bahia, Brazil, **999**

K.